U.S. Department of Labor

Office of Administrative Law Judges
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Issue Date: 25 March 2003

CASE NO.: 2001-BLA-1003

MARTHA C. WILLIAMS, WIDOW OF
ALEXANDER WILLIAMS
Claimant

v.

CONSOLIDATION COAL COMPANY
Employer

and

DIRECTOR, OFFICE OF WORKERS'
COMPENSATION PROGRAMS
Party-in-Interest

Appearances:

Roger D. Forman, Esquire
For the Claimant

Kathy Snyder, Esquire
Dorrie Clark, Esquire
For the Employer

Before: Michael P. Lesniak
Administrative Law Judge

DECISION AND ORDER – AWARDING BENEFITS

This case arises from a claim for benefits under the "Black Lung Benefits Act," Title IV of the Federal Coal Mine Health and Safety Act of 1969, as amended, 30 U.S.C. § 901 et seq. (hereinafter referred to as "the Act"), and applicable federal regulations, mainly 20 C.F.R. Parts 410, 718 and 727 ("Regulations").

Benefits under the Act are awarded to persons who are totally disabled within the meaning of the Act due to pneumoconiosis or to the survivors of persons whose death was caused by pneumoconiosis. Pneumoconiosis is a dust disease of the lung arising from coal mine employment and is commonly known as black lung.[1]

A formal hearing was conducted in Beckley, West Virginia on October 9, 2002, at which time all parties were afforded a full opportunity to present evidence and argument, as provided in the Act and Regulations issued thereunder, found in Title 20, Code of Federal Regulations.[2] The record was left open for the submission of additional medical evidence. Post-hearing, on October 15, 2002, Employer filed Exhibits 1 through 15. They are hereby admitted into evidence as Employer Exhibits 1 through 15. On October 23, 2002, the Employer filed Exhibit 16, a supplemental report of Dr. Zaldivar dated October 7, 2002. On November 6, 2002 the Employer filed Exhibit 17, the supplemental report of Dr. Castle dated November 4, 2002. Counsel for Claimant objected to the submission of these two supplemental reports at the hearing and by letter of October 28, 2002. I find that Employer Exhibits 16 and 17 were submitted as rebuttal to the report of Dr. Cohen. As pointed out by Employer, Dr. Cohen's report was submitted in violation of the 20 day rule (Dr. Cohen's report was dated September 20, 2002 and the hearing was on October 9, 2002). The Regulations specifically provide that an ALJ may keep the record open to allow the submission of post-hearing evidence to respond to evidence submitted in violation of the 20 day rule. 20 C.F.R. § 725.456 (b)(2). Moreover, I find that these additional reports are probative of the issues presented in this matter and are not merely cumulative. *See Lee v. Drummond Coal Co.*, 6 BLR 1-544 (1983). Accordingly, in light of due process considerations and the probative nature of the reports, Claimant's objection to the submission of said evidence is overruled and Employer's Exhibits 16 and 17 are hereby admitted into evidence. The parties filed a "Stipulation of Objective Evidence" on December 20, 2002. Claimant filed her closing argument on January 2, 2003. Employer filed their closing brief on January 15, 2003.

ISSUES

The contested issues are:

1. Whether Claimant has any dependents for purposes of augmentation of benefits;
2. Whether the miner had pneumoconiosis;
3. Whether the pneumoconiosis arose out of the miner's coal mine employment; and
4. Whether the miner's death was due to pneumoconiosis (TR 13)

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[1] The following abbreviations have been used in this opinion: DX = Director's exhibit, EX = Employer's exhibit, CX = Claimant's exhibit, TR = Transcript of the hearing, BCR = Board-certified radiologist, BCI = Board-certified internist, and B = B reader.

[2] At the hearing Director's Exhibits 1-30 and Claimant's Exhibits 1-2 were admitted into evidence without objection. (TR 5, 7).

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Procedural History and Factual Background[3]

The miner, Alexander Williams, filed his first claim for Black Lung benefits on March 30, 1989. (DX 27-1). On August 9, 1989 the Department of Labor denied the miner's claim for benefits. (DX 27-19). On December 1, 1989 the case was referred to the Office of Administrative Law Judges for formal hearing. (DX 27-25). A hearing was held before Administrative Law Judge Joel R. Williams on May 14, 1991. (DX 27-30). On February 5, 1992, Judge Williams issued a Decision and Order Awarding Benefits. (DX 27-31). In that decision, Judge Williams found the miner had established 23 and ¾ years of underground coal mine employment and that he had a one pack per day history of smoking cigarettes for 30 years and continuing. (DX 27-31). Employer appealed the decision to the Benefits Review Board ("BRB"). (DX 27-34). On July 29, 1993, the Benefits Review Board issued a Decision and Order affirming the award of benefits. (DX 27-54). In that decision, the BRB affirmed Judge Williams's finding of pneumoconiosis and his conclusion the miner was totally disabled due to pneumoconiosis. (DX 27-54). Employer did not appeal; therefore, this decision became final. (DX 27-55).

Claimant, Martha Williams, filed her claim for survivor benefits on May 25, 2000. (TR 13, DX 1). The miner died on May 5, 2000. (TR 13). On November 21, 2000, a Notice of Initial Finding was issued finding Employer liable for benefits under the Act. (DX 22). Employer disagreed with the findings and requested a formal hearing. (DX 28).

A formal hearing was scheduled for March 26, 2002 before Administrative Law Judge Gerald M. Tierney but was continued at the request of Claimant.

A hearing was held on October 9, 2002 in Beckley, West Virginia. At that time Claimant testified she was the widow of Alexander Williams and that they had been married for forty-seven years. (TR 15). Claimant has not remarried. (TR 17). She added that the miner had been sick for ten to twelve years before his death. (TR 16). Claimant noted that her husband was on oxygen for several years prior to his death. (TR 16). Claimant testified they adopted their grandson who had been living with them. (TR 16). The miner attended the adoption hearing in his wheelchair. (TR 16). Claimant testified that the miner supported their adopted grandson. (TR 17). The grandson was seventeen and in high school. He did not receive any support from his natural parents. (TR 18).

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[3]Given the filing date of this claim, subsequent to the effective date of the permanent criteria of Part 718, (i.e. March 31, 1980), the regulations set forth at 20 C.F.R. Part 718 will govern its adjudication. Because the miner's last exposure to coal mine dust occurred in West Virginia, this claim arises within the territorial jurisdiction of the United States Court of Appeals for the Fourth Circuit. *See Broyles v. Director, OWCP*, 143 F.3d 1348, 21 BLR 2-369 (10th Cir. 1998).

Medical Evidence

Chest X-rays [4]

X-ray	Reading	Exhibit	Physician	Interpretation
10/20/86	3/27/91	DX 18	Duncan BCR,B	Negative for CWP
10/20/86	3/29/91	EX 4	Gogineni BCR,B	Negative for CWP
10/20/86	4/2/91	EX 4	Abramowitz BCR,B	Negative
10/20/86	5/4/01	DX 18	Wiot BCR,B	Negative for CWP
10/20/86	11/22/01	EX 5	Meyer BCR,B	Negative for CWP
10/20/86	1/15/02	EX 5	Perme BCR,B	Negative
10/20/86	8/25/02	EX 8	Rosenberg B	Negative for CWP
1/18/87	1/18/87	EX 4	Thompson	No active inflammation, no change since 11/20/86
1/18/87	2/25/90	EX 2	Zaldivar B	Negative for CWP
1/18/87	2/7/91	EX 4	Abramowitz BCR,B	0/1, s/t, em
1/18/87	2/11/91	EX 4	Duncan BCR,B	Negative for CWP;em
1/18/87	2/15/91	EX 4	Binns BCR,B	Negative for CWP;em
12/3/87	12/5/87	DX 27	Navarro	1/0, p
12/3/87	2/26/90	EX 2	Zaldivar B	Negative for CWP;fr
12/3/87	2/4/91	EX 4	Shipley BCR,B	Negative for CWP;fr
12/3/87	2/9/91	EX 4	Wiot BCR,B	Negative
12/3/87	2/15/91	EX 4	Spitz BCR,B	Negative for CWP;em

[4] A- A-reader; B- B-reader; BCR- Board-Certified Radiologist; R- Radiologist; BCP- Board-Certified Pulmonologist; BCI Board-Certified Internal Medicine; BCCC-Board-Certified Critical Care. Readers who are board-certified radiologists and/ or B-readers are classified as the most qualified. B-readers need not be radiologists. The existence of pneumoconiosis may be established by chest x-rays classified as category 1, 2, 3, A, B, or C according to ILO-U/C International Classification of Radiographs. A chest x-ray classified as category 0, including subcategories 0/-, 0/0, 0/1, does not constitute evidence of pneumoconiosis. 20 C.F.R. § 718.102(b).

X-ray	Reading	Exhibit	Physician	Interpretation
10/12/88	10/13/88	DX 27-3	Walker[5] Revercomb, or Kugel	Moderate nodular fibrosis, the result of occupational pneumoconiosis
10/12/88	3/--/91	EX 4	Shipley BCR,B	Negative for CWP;fr
10/12/88	3/8/91	EX 4	Wershba BCR,B	Negative
10/12/88	3/11/91	EX 4	Abramowitz BCR,B	Negative
10/12/88	3/13/91	EX 4	Binns BCR,B	Negative
10/12/88	3/14/91	EX 4	Gogineni BCR,B	Negative
10/12/88	3/29/91	EX 4	Spitz BCR,B	Negative for CWP;fr
10/12/88	5/4/01	DX 18	Wiot BCR,B	Negative for CWP;od
10/12/88	11/22/01	EX 5	Meyer BCR,B	Negative for CWP;od
10/12/88	1/15/02	EX 5	Perme BCR,B	Negative
10/12/88	8/25/02	EX 8	Rosenberg B	Negative for CWP;fr
4/26/89	4/26/89	DX 27	Daniel	0/0
4/26/89	5/19/89	DX 27	Zaldivar B	Negative;em;fr
4/26/89	2/8/91	EX 4	Scott BCR,B	Negative for CWP;em fr;comments
4/26/89	2/8/89	EX 4	Wheeler BCR,B	Negative for CWP;fr
4/26/89	3/4/91	EX 4	Abramowitz BCR,B	0/1, s/t;comments
4/26/89	3/6/91	EX 4	Duncan BCR,B	Negative for CWP;em fr;comments
4/26/89	3/14/91	EX 4	Gogineni BCR,B	Negative for CWP;em fr
4/26/89	3/19/91	EX 4	Wershba BCR,B	Negative for CWP;fr
4/26/89	3/28/91	EX 4	Shipley BCR,B	Negative for CWP;fr
4/26/89	3/29/91	EX 4	Spitz BCR,B	Completely negative
1/24/90	2/25/90	EX 2	Zaldivar B	Negative for CWP;fr
1/24/90	2/5/91	EX 4	Scott BCR,B	Negative for CWP;fr comments
1/24/90	2/5/91	EX 4	Wheeler BCR,B	Negative for CWP;fr comments

[5] This x-ray was referenced in the October 13, 1988 findings of the West Virginia Occupational Pneumoconiosis Board. The Board's report indicated that the x-ray revealed moderate, nodular fibrosis, "the result of occupational pneumoconiosis." (DX 27-3). Although the actual reader is not specified, the Board's report stated that the films relied upon were made by "a member of the Board."

X-ray	Reading	Exhibit	Physician	Interpretation
1/24/90	3/4/91	EX 4	Abramowitz BCR,B	0/1, s/t;comments
1/24/90	3/6/91	EX 4	Duncan BCR,B	Negative for CWP;fr
1/24/90	3/14/91	EX 4	Gogineni BCR,B	Negative for CWP;em fr
1/24/90	3/19/91	EX 4	Wershba BCR,B	Negative for CWP;fr; comments
1/24/90	4/24/01	DX 18	Wiot BCR,B	Negative for CWP;od comments
1/24/90	3/28/91	EX 4	Shipley BCR,B	Negative for CWP;fr; comments
1/24/90	3/29/91	EX 4	Spitz BCR,B	Negative;fr
1/24/90	11/22/01	EX 5	Meyer BCR,B	Negative for CWP;od; comments
1/24/90	1/15/02	EX 5	Perme BCR,B	Negative for CWP; comments
1/24/90	8/28/02	EX 8	Rosenberg B	Negative for CWP;em fr; comments
12/26/90[6]	12/26/90	------	Aycoth B	1/1, p/q, 6 zones
12/26/90	1/8/91	------	Ahmed B	1/1, p/p, 6 zones
12/26/90	2/8/91	------	Pathak B	2/2, p/q, 6 zones
12/26/90	12/26/90	------	Cappiello B	1/2, p/q, 6 zones
12/26/90	3/20/91	EX 4	Scott BCR,B	Negative for CWP;fr; comments
12/26/90	3/20/91	EX 4	Wheeler BCR,B	Negative for CWP;fr; comments
3/15/98	3/16/98	DX 15	Dehgan	Portable. Right upper lobe and rt lung base lesions
3/21/98	3/21/98	DX 15	Patel	No internal change in right pneumothorax. Mass in rt upper lobe

[6] The x-ray interpretation reports of Drs. Aycoth, Ahmed, Pathak, and Cappiello of the December 26, 1990 chest x-ray are missing from the current case file. However, they are described by Judge Williams in the January 27, 1992 Decision and Order, page 4 (DX 27-31) and, in detail, in the miner's post-hearing brief dated September 10, 1991, page 3-4 (DX 27-29). Judge Williams noted that this x-ray was read as positive for pneumoconiosis by the foregoing physicians and that all of these physicians were B-readers of chest x-rays. This finding was confirmed in the decision issued by the Benefits Review Board. (DX 27-54).

X-ray	Reading	Exhibit	Physician	Interpretation
3/22/98	3/23/98	DX 15	Patel	Internal re-expansion of rt lung w/resolution of pneumothorax
3/23/98	3/23/98	DX 15	Patel	Interval resolution of pneumothorax;
3/25/98	3/25/98	DX 15	Patel	No pneumothorax seen. COPD.
3/25/98	8/25/02	EX 8	Rosenberg B	u/r; comments

CT Scan Reports

A CT scan was performed on March 18, 1998. The scan was read by Dr. Patel as showing a 2.0 cm ill-defined mass in the right upper lobe, posteriorly. (DX 15). The scan was subsequently read by Dr. Wiot on April 24, 2001 as showing no evidence of coal worker's pneumoconiosis ("CWP") and that the lung fields showed mild emphysemateous change and findings consistent with malignancy involving the right upper lobe. (DX 18). The CT scan was also read by Dr. Meyer on November 22, 2001. He indicated the scan showed no evidence of CWP. However, Dr. Meyer did note the presence of a 2.4 cm speculated mass in apical segment of right upper lobe suspicious for bronchogenic carcinoma, a nonspecific nodule in superior segment of right lower lobe, and a neoplastic etiology could not be excluded. (EX 5). The CT scan was read by Dr. Perme on January 15, 2002. He found there were no changes of CWP. In addition, Dr. Perme noted the right upper lobe apical segment had a speculated mass suspicious for lung cancer and the presence of mildly enlarged AP window lymph nodes which may have represented nodal spread of lung cancer. (EX 5).

Occupational Pneumoconiosis Board

On October 13, 1988, the Occupational Pneumoconiosis Board awarded the miner a 50% pulmonary function impairment attributable to coal worker's pneumoconiosis. This represented a 45% increase in impairment from the 5% award in 1986. The members based their decision on a physical examination by the Board, pulmonary function studies by the Board, and an x-ray read by a member of the Board. It was noted that film studies showed a nodular fibrosis in a moderate amount, the result of occupational exposure. (DX 27-3).

Medical Reports[7]

Dr. John Daniel

The medical report of Dr. Daniel is dated April 26, 1989 and appears at DX 27-14. Dr. Daniel reviewed the miner's occupational history and noted a family history of diabetes. He noted a smoking history of one pack of cigarettes per day for 30 years and continuing. He noted that the miner complained of sputum production, wheezing, dyspnea, cough, and orthopnea. Physical examination revealed dry wheezing and rales bilaterally. An x-ray was read as negative for CWP, pulmonary function testing showed a severe obstructive defect, and arterial blood gases were normal. Dr. Daniel diagnosed the miner as having chronic obstructive pulmonary disease ("COPD") due to 30 years of smoking based on a history of chronic productive cough, history of smoking, and evidence of an obstructive defect. He concluded there was no evidence of a significant pulmonary dysfunction.

The supplemental medical report of Dr. Daniel is dated March 25, 1991 and appears at EX 4. Dr. Daniel stated that the miner showed no evidence of significant pulmonary dysfunction and therefore, no evidence of respiratory impairment. Because there was no pulmonary impairment, the miner should have been able to perform his usual coal mine employment. Dr. Daniel also noted that there was no evidence of pneumoconiosis in the examination he performed on the miner.

[7] After my review of the record it appears that documents identified as Claimant's exhibits 1 through 10 and Employer's Exhibits 1 through 12 in the *closed living miner's claim* are missing from the current case file. (see May 14, 1991 hearing transcript admitting said exhibits, DX 27-30, page 8-12). However, most of the missing exhibits appear elsewhere in the record except for the following: medical report of Dr. Renn dated March 27, 1991; medical report of Dr. Castle dated April 18, 1991; deposition of Dr. Zaldivar dated April 23, 1991; medical report of Dr. Rasmussen dated February 6, 1990; pulmonary function study dated October 12, 1989; and the positive x-ray interpretations of an x-ray dated December 26, 1990 from Drs. Aycoth, Ahmed, Pathak, and Cappiello. I will discuss the relevance of this missing evidence in the "Conclusions of Law" section of this opinion.

Dr. George Zaldivar

The first medical report of Dr. Zaldivar is dated February 26, 1990 and appears at EX 2. He examined the miner on January 24, 1990. He concluded the miner was a current smoker based on a high carboxyhemoglobin test, there was no radiographic evidence of CWP, the miner had a moderate reversible airway obstruction, and that the miner had normal arterial blood gases. Dr. Zaldivar concluded the miner had asthma and mild emphysema. He added that the miner did not have CWP based on the miner's history, physical examination, and lab work. He noted the miner's asthma, which was a disease of the general population, was unrelated to coal mine work. Dr. Zaldivar stated the miner had a small amount of emphysema due to his life-long history of smoking.

The second medical report of Dr. Zaldivar is dated July 13, 1991 and appears at EX 2. Basically, this report was a reiteration of the first report dated February 26, 1990.

The supplemental report of Dr. Zaldivar is dated February 28, 1994 and appears at EX 2. He reviewed additional records including a record review by Dr. Fino dated April 24, 1992[8] and a medical report from Dr. Rasmussen dated August 4, 1992. He also reviewed deposition testimony from Dr. Rasmussen taken on August 24, 1993.[9] Dr. Zaldivar concluded that Dr. Rasmussen did not provide any new information that would justify his claim that the miner suffered from CWP rather than asthma and emphysema; asthma as a result of genetic predisposition and emphysema from smoking. He added that his statement that the miner did not have CWP was not based on an x-ray alone and that the miner met all of the requirements for the diagnosis of asthma.

Dr. W.K.C. Morgan

The medical report of Dr. Morgan is dated April 17, 1991 and appears at EX 4. Dr. Morgan reviewed various medical records including the four positive chest x-ray interpretations by Drs. Pathak, Ahmed, Cappiello, and Aycoth of the December 26, 1990 x-ray. Dr. Morgan readily dismissed these readings and stated "I place no reliance on their opinion." Dr. Morgan concluded the miner had moderate obstruction with some reversibility and that his main problem was emphysema. He noted that many subjects that were current smokers with chronic bronchitis and emphysema showed some reversibility. Dr. Morgan opined the miner's airway obstruction was related to cigarette smoking and not to asthma. He also opined that the x-ray was negative for CWP based on several radiologists' interpretations. He noted that despite the fact that the miner spent a fair time underground, there was no evidence of pneumoconiosis. Dr. Morgan stated, "The fact that Mr. Williams has no radiographic evidence of pneumoconiosis is a strong indication that the amount of dust that is present in his lungs is strictly limited and argues cogently against Dr. Rasmussen's thesis." He agreed that the miner may well have been partially disabled by the amount of airways obstruction but that none of his impairments were job related. Instead they were related to naturally occurring disease, to smoking, and the excessive use of alcohol.

[8] This report by Dr. Fino is not part of the record.

[9] This deposition testimony of Dr. Rasmussen is not part of the record.

Dr. D.L. Rasmussen

The medical report of Dr. Rasmussen is dated March 14, 1991 and appears at DX 14. Dr. Rasmussen noted that the miner had been under his care since September 28, 1989. He added that the miner had the following respiratory conditions: CWP, chronic bronchitis, and pulmonary emphysema. He stated the pulmonary function studies performed on October 12, 1989 showed moderate, partially reversible obstructive ventilatory impairment. He noted these studies were consistent with chronic bronchitis and at least mild emphysema. He opined that the degree of pulmonary impairment would clearly render the miner totally disabled from his coal mine employment. In addition to his own observations, Dr. Rasmussen also reviewed the medical reports of Drs. Daniel and Zaldivar, various chest x-ray reports, and his own medical report from 2-6-90. He disagreed with Dr. Daniel's assertion that because the miner's exercise blood gases were found to be within normal range that he had no significant pulmonary impairment. Dr. Rasmussen opined instead that the miner "obviously" had a disabling ventilatory insufficiency which was totally independent of any gas exchange capacity. He added that the miner had two or three possible risk factors for his disabling respiratory insufficiency: 20 years of underground coal mine employment, significant smoking history of 1 and ¼ packs per day for 30 years, and a reported history of bronchial asthma by Dr. Zaldivar. Dr. Rasmussen stated that he disagreed with Dr. Zaldivar's diagnosis of bronchial asthma. He opined that Dr. Zaldivar's finding of partial reversibility of airway obstruction was not, per se, diagnostic of asthma. In fact, Dr. Rasmussen pointed to a study that noted that unless the reversal of airway obstruction was complete, reversibility of airway obstruction was not diagnostic of bronchial asthma since it could occur in any type of COPD. Dr. Rasmussen noted the disparity in the interpretation of chest x-rays for the presence of pneumoconiosis. He added it was well known that pneumoconiosis could be present in significant degree without being visible by x-ray. Moreover, Dr. Rasmussen noted that aside from the presence or absence of coal pneumoconiosis in this case, there was strong evidence the miner's occupational dust exposure was at least a significant contributing factor to his totally disabling respiratory insufficiency. He cited to several medical sources in support of his assertion. Dr. Rasmussen concluded that based on recent medical literature it was impossible to separate the effects of smoking from those of coal mine dust exposure. He added, in the case of the miner, one must conclude that this totally disabling respiratory insufficiency was, at least in part, the consequence of his coal mine dust exposure with its resultant pneumoconiosis as well as COPD.

The second medical report of Dr. Rasmussen is dated August 4, 1992 and appears at DX 14. He reviewed the April 24, 1992 report of Dr. Fino and the July 31, 1991 report of Dr. Zaldivar. He cited to a study that showed that 30% of moderate and 20% of severe macular types of pneumoconiosis and 29% of mild micronodular pneumoconiosis were reported with negative chest x-rays thus indicating the imperfection of the chest x-ray. Dr. Rasmussen disagreed with Dr. Fino's conclusions because Dr. Fino's main reason for discounting the miner's dust exposure as a causative factor in his disabling lung disease was the fact that he had a pure obstructive vent defect as opposed to a restrictive defect. He cited to studies that showed that significant airways obstruction may occur as a result of coal mine dust exposure in the absence of x-ray changes. He pointed to another study that showed both smoking and coal mine dust may produce emphysema

in coal workers. He concluded by stating that he disagreed with the reports of Drs. Zaldivar and Fino and that the medical evidence clearly indicated that coal mine dust exposure was fully capable of producing the type and severity of impairment encountered in the miner. He maintained his opinion that the miner did have CWP which arose from his coal mine employment and that the miner's coal mine dust exposure was at least a major contributing factor to his disabling respiratory insufficiency.

Miscellaneous Medical Records

The record contains the medical records from the Clear Fork Clinic from 1982 through 1998. (DX 27). The record also contains the medical notes from the Southern WV Clinic from September 28, 1989 through March 15, 2000. (DX 14). The record also contains the medical records, including several admission and discharge summaries, from Appalachian Regional Healthcare, Inc.. (DX 15).

Death Certificate

The death certificate is dated May 9, 2000 and appears at DX 13. The miner's date of birth was noted as November 20, 1929. The date of the miner's death was noted as May 5, 2000. The immediate causes of death were noted as advanced carcinoma of the lung and respiratory failure. The certifying physician was Dr. Velayudhan Sahadevan.

Autopsy Report

No autopsy was performed in this matter.

Post-Mortem Medical Reports

Dr. Dominic Gaziano

The medical report of Dr. Gaziano is dated August 31, 2000 and appears at DX 16 of the record. Dr. Gaziano is Board-Certified in Internal Medicine and Chest Disease and is a certified B-reader of chest x-rays. (DX 16). Dr. Gaziano conducted a review of the medical records at the request of the Department of Labor. Dr. Gaziano opined that the miner had coal worker's pneumoconiosis and that it was a substantially contributing cause or factor leading to the miner's death. He noted that the right lung biopsy of May 10, 1999 was strongly suggestive of the presence of CWP. He added that the miner died of lung cancer that had been found at an early stage, however, curative surgery was thwarted by the presence of severe lung disease (emphysema and CWP).

Dr. Stephen T. Bush

The medical report of Dr. Bush is dated May 2, 2001 and appears at DX 17 in the record. Dr. Bush is Board-Certified in Anatomical and Clinical Pathology. (DX 17). Dr. Bush reviewed, among many records, the miner's occupational history, various medical records from ARH Hospital and Southern West Virginia Clinic, letters from Dr. Rasmussen, various chest x-ray reports, a right lung biopsy report, a head CT scan report, death certificate, and one histological slide. He noted a smoking history of 55 pack years. A microscopic review of the single histologic slide showed, in his opinion, four tiny fragments of bronchial muscosa that showed only a small amount of adjacent lung tissue. Dr. Bush noted the presence of a very small amount of fine dust particles in small aggregates, widely scattered. He stated, "The identity of these particles is uncertain and might be the result of tobacco or inhaled coal dust." Dr. Bush concluded that the biopsy tissue was too limited with an inadequate amount of lung tissue to either confirm or deny the presence of CWP. Dr. Bush agreed with Dr. Rasmussen's opinion that CWP may be present in the absence of positive x-ray changes. But then stated, "In my experience, this does occur but the degree of coal worker's disease in the circumstances of multiple x-rays and CT scans negative for evidence of coal worker's pneumoconiosis, such as in Mr. Williams, results in autopsy findings of a very limited degree of disease. For this reason, I cannot make a diagnosis of a significant degree of coal worker's pneumoconiosis with reasonable medical certainty." Dr. Bush also concluded that coal worker's disease did not contribute to the death of the miner. He stated that there was no causal relationship between coal mine dust and the development of lung cancer. Moreover, he criticized the assertion of others that coal worker's disease contributed to a pulmonary impairment which prevented curative surgery as being "presumptuous" and "beyond reasonable medical certainty." He added that the surgical cure rate for this type of cancer was "very poor" and that radiation treatment was equally as effective. Dr. Bush acknowledged that the miner suffered from severe pulmonary impairment as a result of emphysema. He then noted the miner's 55 pack year history of smoking. He stated that the miner was totally disabled prior to death due to carcinoma and its complications, severe pulmonary emphysema, diabetes, and hypertensive cardiovascular disease. Dr. Bush concluded, "The coal worker's pneumoconiosis from all available evidence was too mild in degree and extent to have contributed to impairment or death." Dr. Bush then went on to critique studies referenced by Dr. Rasmussen then concluded, "I am not persuaded that Dr. Rasmussen has provided information that causally links coal mine dust exposure to significant pulmonary emphysema in the absence of significant coal worker's pneumoconiosis."

The supplemental report of Dr. Bush is dated March 4, 2002 and appears at EX 6. Dr. Bush reviewed additional medical records, various chest x-ray interpretations, the consultation reports of Drs. Naeye, Caffrey, and Zaldivar, five additional histologic slides from a right lung biopsy labeled "C99-4017" and another slide labeled "S98-01393". Regarding these slides, Dr. Bush noted a moderate amount of dust pigment but added that the tissue fragments were too small to evaluate the presence or absence of CWP. Dr. Bush concluded the lungs showed no evidence of CWP based on abundant radiographic evidence that indicated the absence of changes for "significant coal worker's pneumoconiosis." He opined that CWP did not contribute to the death of the miner. The miner died as a result of metastatic squamous cell carcinoma that was causally associated with heavy cigarette smoking. Dr. Bush acknowledged the miner suffered from severe chronic obstructive pulmonary disease that became progressively more severe as smoking

continued. He opined that CWP and occupational exposure to coal dust did not contribute in any way to respiratory impairment. Dr. Bush stated that CWP, if present, was too limited in degree and extent to have contributed to respiratory impairment. Moreover, he added that death would have occurred at the same time and in the same manner from the complications of carcinoma of the lung resulting from a long history of cigarette smoking if the miner had never been exposed to the pulmonary hazards of coal mining employment. Dr. Bush included some additional comments regarding the conclusions reached by Drs. Rasmussen and Gaziano.

The second supplemental report of Dr. Bush is dated August 29, 2002 and appears at EX 8. Dr. Bush reviewed additional x-ray interpretations and chest CT scan reports from Drs. Wiot, Meyer, and Perme of a CT scan taken on March 18, 1998. Dr. Bush also reviewed portions of the ALJ decision and order that awarded benefits during the miner's lifetime. Dr. Bush clarified that although the needle biopsies produced insufficient evidence to confirm or deny the presence of CWP, he did conclude from other medical information that significant CWP sufficient to cause impairment or contribute to death was not present. He noted that the absence of significant CWP was supported by the absence of abnormal arterial blood gases, the absence of evidence of cor pulmonale, and the absence of consistent radiologic changes as noted in the x-ray reports. He disagreed with the ALJ's findings and stated, "I am not convinced by the judge's decision that significant coal worker's pneumoconiosis was present or contributed to disability or death….The explanation for the judge's conclusions focuses on an aberrant, old (12/26/90) x-ray report and somewhat convoluted interpretations of the findings of Dr. Renn, Dr. Morgan and Dr. Zaldivar regarding the evalution [sic] of pulmonary function study results."

The third supplemental report of Dr. Bush is dated October 2, 2002 and appears at EX 15. After reviewing Dr. Cohen's report, Dr. Bush concluded that the miner died from complications of tobacco addiction: chronic obstructive pulmonary disease and carcinoma of the lung. He added that there was no objective evidence that suggested that coal dust exposure played a significant role in the events leading to death. Dr. Bush noted there was no objective evidence that supported the diagnosis of simple CWP. He concluded the records provided a high degree of medical certainty that death would have occurred at the same time and in the same manner if the miner had never been exposed to the pulmonary hazards of coal mining employment.

Dr. Richard L. Naeye

The medical report of Dr. Naeye is dated June 9, 2001 and appears at DX 19 in the record. Dr. Naeye is Board-Certified in Anatomic and Clinical Pathology. (DX 19). Dr. Naeye reviewed an occupational history, various medical records from St. Francis Hospital, The Southern West Virginia Clinic, Appalachian Regional Hospital, the reports of Drs. Wiot, Zaldivar, Rasmussen, Sandler, Maramba, Patel, Imbing, and Bou-Abboud, and the consultation reports of Drs. Bush and Rasmussen. Dr. Naeye noted an occupational history of 23.3 years of underground coal mine employment ending in 1989. He also noted a smoking history of 45 pack years that ended in 1995. He reviewed a pulmonary function study from October of 1989 that suggested the presence of chronic bronchitis and mild emphysema. Dr. Naeye reviewed the single histologic

slide containing four tiny pieces of bronchial wall. He noted that there were no neoplastic cells or findings of CWP in the small tissue samples. Dr. Naeye opined, "Without conclusive evidence of coal workers' pneumoconiosis (CWP) and its severity it is most difficult to ascertain if it had a role in his death." He noted that although most of the chest x-rays were negative for CWP, this did not exclude its presence in a mild form. He noted lesions of CWP could progress after leaving the industry if a substantial amount of free silica was present in the CWP lesions. He noted there was no free silica in the small sample he was provided. Dr. Naeye cited to studies that concluded there was no connection between mining coal and the development of lung cancer. Dr. Naeye noted the presence of a moderate severe airway obstruction and concluded if the miner had been a non-smoker for most of his adult life, it was "unlikely" he would have developed this airway obstruction. He reasoned that cigarette smoking had a three- to- four time greater influence in causing airway obstruction in coal miners than long time exposure to coal mine dust. Dr. Naeye also cited to studies that showed bronchitis and bronchioloitis, the main causes of airway obstruction, have little or no effect on lung function unless the subject happens to be a smoker. He also noted, "Airway obstruction caused by centrilobular emphysema and bronchitis that is severe enough to preclude a miner from working is very rare if it indeed occurs at all in the absence of smoking or complicated CWP."(citations to studies omitted). Dr. Naeye then opined that based on this information "it is unreasonable to postulate that CWP had a significant role in causing disability or hastening the death of Alexander Williams."

The supplemental report of Dr. Naeye is dated February 17, 2002 and appears at EX 6. Dr. Naeye reviewed the consultation reports of Drs. Bush, Caffrey, Castle, and Meyer. He also reviewed various additional x-ray reports that were all interpreted as negative for CWP. Dr. Naeye also received arterial blood gas studies from March 15, 1998 and April 25, 1998 and additional medical records from various clinics. He also received one histologic slide labeled "S98-01393" and five glass slides containing tissue taken from a biopsy of the miner's right upper lobe labeled "C99-04017". He noted the presence of old fibrous tissue with a moderate amount of black pigment and a few moderate sized birefringent crystals of non-toxic silicates. Dr. Naeye stated that two types of findings needed to be present to make a diagnosis of CWP. First, anthracotic pigment must be identified adjacent to small arteries and airways and beneath the pleura in the lungs. Second, tissue damage must be associated with the pigment. He concluded that the tissue provided did not have clearly identifiable anthracotic macules or larger black deposits around which focal emphysema could be identified. Thus, the available tissue findings did not support the diagnosis of CWP. He then went on to add that if CWP were present, it was too mild to have caused clinically significant abnormalities in lung function and thus could not have hastened the miner's death. He reasoned that CWP macules, micronodules, and macronodules, when plentiful enough to impair lung function were easily seen on x-rays and CT scans. He noted that these lesions were not routinely visible in images of the miner's lungs. He added that this did not mean that CWP lesions were completely absent from his lungs. Dr. Naeye concluded, "If any were present they were too small in size and few in number to have caused disability or hastened death."

The second supplemental report of Dr. Naeye is dated September 27, 2002 and appears at EX 11. Dr. Naeye reviewed the reports of Drs. Bush, Rosenberg, Zaldivar, and Cohen. Dr. Naeye disagreed with Dr. Cohen's conclusion that the miner's "pulmonary insufficiency and death were in large part the result of his occupational exposures to coal mine dust." Dr. Naeye cited to a study that showed that the effects of smoking cigarettes were x4.5 greater than the effects of mine dust. His analysis of background literature suggested that mine dust played no role or only a minor role in the genesis of centrilobular emphysema. Moreover, he also concluded that there was "strong evidence that simple CWP does not progress after a miner quits the industry and leaves the exposure to coal mine dust." Dr. Naeye also criticized Dr. Cohen's reliance on studies performed in the United Kingdom, Sardinia, and Germany. Dr. Naeye concluded that cigarette smoking, not exposure to coal mine dust, was the major cause of pulmonary disability and resulting death in US bituminous coal miners.

Dr. P. Raphael Caffrey

The medical report of Dr. Caffrey is dated June 27, 2001 and appears at EX 1 of the record. Dr. Caffrey is Board-Certified in Anatomical and Clinical Pathology. (EX 1). Dr. Caffrey reviewed the miner's employment history, various reports and records of Dr. Rasmussen, various hospital records, the consultation reports of Drs. Sahadevan and Bou-Abboud, a cytology report from May 10, 1999, a pathology report from March 27, 1998, copies of various chest x-ray reports, the miner's death certificate, the medical opinions of Drs. Gaziano, Bush, and Naeye, and one surgical pathology slide labeled "S98-01541". Dr. Caffrey conducted a microscopic review of the single pathology slide that included biopsy fragments of the right upper lobe. The accompanying report was dated March 27, 1998. Dr. Caffrey noted there were "few very fine black particles in small aggregates, and these small, fine, black particles do not appear typical for inhaled coal dust….but obviously I cannot exclude coal dust particles." He saw no evidence of fibrosis. Dr. Caffrey noted a 20 year history of coal mine employment. He also discussed a varied smoking history that ranged from 30 to 55 pack years, depending on the report. Dr. Caffrey reviewed the miner's medical history and concluded that he could not make a diagnosis of CWP but he could not exclude simple CWP as being present. He added that the "biopsies were not adequate for me to be able to make that statement." Dr. Caffrey concluded that the amount of cigarette smoking the miner did over the years was the principle cause of the following diseases: carcinoma of the lung, emphysema, hypertensive cardiovascular disease, esophageal reflux, and chronic bronchitis. Dr. Caffrey stated that assuming the miner had CWP, the degree of simple CWP would have been mild and "certainly would not have caused him [the miner] the COPD which he suffered from." In support of this position, Dr. Caffrey recapped the results of several studies that concluded: (1) cigarette smoking is the major risk factor associated with the development of COPD, (2) the inhalation of coal dust in the absence of smoking and complicated pneumoconiosis rarely induces sufficient ventilatory impairment to preclude a miner from working, (3) most miners with simple CWP have no symptoms or physical signs, and (4) miners with simple CWP have demonstrated several minor abnormalities in pulmonary function but that these abnormalities were "most noticeable" in miners with higher categories of simple CWP and were also found in non-smoking miners. In summary of his position, Dr. Caffrey stated that he could not rule out the possibility the miner may have had CWP. Assuming the miner had CWP, he opined that it would have been mild as evidenced by most of the negative chest x-rays and "would not, by itself, have caused him pulmonary disability and caused him to retire from the

mines." He disagreed with Dr. Gaziano's opinion that pneumoconiosis was a substantially contributing factor to the miner's death. Dr. Caffrey noted that had the miner been a non-smoker, he would not have developed carcinoma of the lung, significant COPD, and atherosclerotic heart disease and would not have had to retire at age 59. He opined that if the miner had simple CWP, it would not have caused him disabling pulmonary impairment and would not have caused or hastened his death.

The supplemental report of Dr. Caffrey is dated March 18, 2002 and appears at EX 6. Dr. Caffrey reviewed additional medical evidence including the consultation reports of Drs. Zaldivar and Castle, various chest x-ray interpretations, two biopsy reports dated March 20, 1989 and May 7, 1999, one pathology slide labeled "S98-01393" and five slides labeled "C99-4017; R Lung Bx," and the miner's death certificate. Microscopic examination of slide "S98-01393" showed clusters of neoplastic squamous cells along with moderate amounts of anthracotic pigment but no true macules were identified. Microscopic examination of the five slides labeled "C99-4017" showed numerous sheets of malignant squamous cells along with a moderate amount of anthracotic pigment, but no macules were seen. Dr. Caffrey noted that these samples did not contain enough tissue to rule out the presence of CWP. The additional medical evidence presented to Dr. Caffrey did not change his opinions rendered in his initial report. In summary, Dr. Caffrey concluded that the miner suffered from a totally disabling pulmonary respiratory disability prior to death due to years of smoking cigarettes which caused his emphysema and carcinoma of the lung. He also concluded the coal dust did not cause the miner disability prior to his death, nor did it cause, contribute to, or hasten the miner's death. Assuming the miner had CWP, Dr. Caffery opined this simple CWP would have been so mild it would not by itself have caused pulmonary disability.

The second supplemental report of Dr. Caffrey was dated October 2, 2002 and appears at EX 14. Dr. Caffrey reviewed the medical report of Dr. Cohen and concluded, based on the documents he received, there was no evidence the miner "definitely" suffered from CWP. He then stated the miner "may have had simple CWP…but a diagnosis of CWP was never absolutely established so I definitely do not agree with Dr. Cohen's statement [i.e. the miner did indeed suffer from CWP]." Dr. Cafrrey noted it was possible for miners to have simple CWP even when x-rays were read as negative for CWP. However, it was his experience that when he actually reviewed the lung findings at autopsy, those miners only had a mild degree of simple CWP; a degree of simple CWP that would not have disabled the patient. Dr. Caffrey strongly disagreed with Dr. Cohen's statement that coal dust and tobacco smoke produced similar decrements in lung function. Moreover, Dr. Caffrey stated that the miner was a poor candidate for surgery due to his lung condition and his cardiac condition (coronary artery disease). Therefore, Dr. Caffrey concluded that even if the miner's only medical condition was a mild degree of simple CWP, this mild degree of simple CWP certainly would not have prevented him from having surgery for his lung cancer.

Dr. George L. Zaldivar

The medical report of Dr. Zaldivar is dated September 4, 2001 and is found at EX 3 in the record. Dr. Zaldivar is Board-Certified in Internal Medicine, Pulmonary Disease, and Critical Care Medicine. He is also a certified B-reader of chest x-rays. (EX 2). Dr. Zaldivar examined the

miner on several previous occasions. Dr. Zaldivar reviewed his reports of July 13, 1991 and February 28, 1994. He also reviewed the medical notes of Dr. Rasmussen, medical records from Appalachian Regional Medical Center, various blood gas reports, various chest x-ray interpretations, a pathology report of a biopsy on March 7, 1999, a CT scan report of March 18, 1998, the miner's death certificate, a medical report of Dr. Gaziano, biopsy reports of a biopsy done on May 7, 2000, and the consultation reports of Drs. Bush, Naeye, and Caffrey. Dr. Zalidvar noted a history of 26 years of coal mine employment and a smoking history of 1 and ¼ packs per day from the age of 17. Based on all of this information and his own examination of the miner, Dr. Zaldivar concluded there was no evidence of coal worker's pneumoconiosis or any dust disease of the lungs. He added there was a significant respiratory impairment present in life and that it was due to a combination of asthma and emphysema caused by the miner's lifelong history of intensive smoking. Dr. Zaldivar added, from a pulmonary standpoint prior to death, the miner was unable to perform any work due to the severity of the pulmonary disease brought about by asthma and emphysema. He noted the miner had coronary artery disease that played some role in the shortness of breath. The coronary artery disease ("CAD"), itself, may have been incapacitating, preventing the miner from performing any work. Dr. Zaldivar concluded that coal dust exposure did not play any role in the miner's death. He added that even if the miner had early simple CWP, his opinion regarding the cause of death would not have changed at all. Dr. Zaldivar opined the miner died as a result of all of the terrible effects that cancer produces in a human: anemia, weight loss, and rib fractures. None of these problems were related to his occupation as a coal miner. Dr. Zaldivar noted emphysema was an added factor, but not the primary cause of death. He stated that smoking was responsible for the lung cancer. Dr. Zaldivar further concluded that even if the miner had never worked in the coal mines, he would have died when and as he did, as a result of cancer of the lung.

The supplemental report of Dr. Zaldivar is dated August 22, 2002 and appears at EX 8. Dr. Zaldivar reviewed additional information including additional chest x-ray findings and CT scans. He noted that the biopsies of record were those of the cancer itself and not of the lung and therefore there was inadequate tissue to determine whether or not pneumoconiosis was present. Dr. Zaldivar went into detail regarding the advantages of using a CT scan for diagnosing CWP. Dr. Zaldivar concluded that based on the conventional (as opposed to a high resolution) CT scan and chest x-rays, the miner did not have CWP. He added that smoking lead to the occurrence of cancer in the lung which in turn caused the miner's death. He noted this smoking habit caused some degree of emphysema and that there was a co-existing asthma which was contributing to the COPD from which the miner suffered. Dr. Zaldivar concluded the miner did not have CWP nor did he die as a result of CWP, whether directly or indirectly. He added that the cause of death was lung cancer and that the pulmonary impairment in life was due to smoking and asthma, unrelated to pneumoconiosis.

The second supplemental report of Dr. Zaldivar is dated October 7, 2002 and appears at EX 16. After carefully analyzing the report of Dr. Cohen, Dr. Zaldivar concluded there was no question that the miner may have had a better prognosis had surgery been performed. Moreover, there was no question that surgery could not be performed because of the poor condition of the miner's lungs, not to mention the presence of coronary artery disease. However, Dr. Zaldivar concluded that death was not due to the miner's lungs but was due to cancer itself. Dr. Zaldivar added that "[M]y opinion remains….that Mr. Williams did not have coal worker's

pneumoconiosis." He noted that he had diagnosed asthma in 1988 and that the miner developed severe emphysema on top of his asthmatic substrate.

Dr. James Castle

The medical report of Dr. Castle is dated January 30, 2002 and appears at EX 7. Dr. Castle is Board-Certified in Internal Medicine and Pulmonary Disease and is a certified B-reader of chest x-rays. Dr. Castle reviewed the miner's occupational history and noted 25 years of coal mine employment. He reviewed various medical records and noted a smoking history of one pack of cigarettes per day from the age of 20 and continuing up through at least 1989. He reviewed many x-ray interpretations, outpatient and hospital records, and the medical reports of Drs. Daniel, Morgan, Rasmussen, Gaziano, Bush, Naeye, Caffrey, and Zaldivar. Dr. Castle concluded the miner did not suffer from CWP and that the miner's death was not caused by, contributed to, or hastened in any way by CWP or coal mine dust exposure. He acknowledged the miner had sufficient exposure to coal dust to develop CWP if he were a susceptible host and that he also had a sufficient smoking history to develop chronic obstructive pulmonary disease in a susceptible host. After noting that the vast majority of the x-ray interpretations were negative for CWP, Dr. Castle concluded there was no radiographic evidence of CWP. He did acknowledge it was possible to have pathologic evidence of CWP without radiographic evidence, but in that case, the actual amount of dust in the lung was generally insufficient to cause significant impairment or disability. Based on his review of the medical histories, physical examinations, radiographic evaluations, and physiologic testing, Dr. Castle concluded the pulmonary impairment that was present was not due to CWP nor was it due to his coal mine dust exposure. In summary, Dr. Castle concluded the miner did not have CWP. He opined that the miner did not have the physical findings, radiographic findings, pathological findings, or the physiological findings to indicate the presence of that disease process. Dr. Castle opined further that the miner was permanently and totally disabled due to tobacco- smoke- induced pulmonary emphysema and bronchial asthma prior to his death. He added the miner did not suffer from a chronic disease of the lungs that was caused by, contributed to, or substantially aggravated by coal mine dust exposure. Moreover, the miner's death was not caused by, contributed to, or hastened in any way by CWP or his coal mine dust exposure. It was his opinion that the miner would have died as and when he did regardless of his occupational history.

The supplemental report of Dr. Castle is dated September 5, 2002 and appears EX 8. Dr. Castle reviewed additional x-ray interpretations and chest CT scan reports from Drs. Meyer, Perme, and Wiot. Dr. Castle noted the CT scan offered a better means of evaluation of chest disease and determination of parenchymal abnormalities than did a standard chest x-ray. He opined, since this imaging device was more sensitive, it was therefore more accurate for determination of parenchymal changes such as those seen with CWP. He noted that none of those changes were found in this case. Dr. Castle added that even if the miner had been found to have simple CWP, his opinions concerning the cause of death would remain unchanged. He concluded the miner's death was due to complications of bronchogenic carcinoma caused by tobacco smoking. Dr. Castle added that the lung cancer ultimately developed into a Pancoast syndrome with resultant chest wall and other involvement.

The deposition of Dr. Castle was taken on October 1, 2002 and appears at EX 10. Dr. Castle reiterated there was no radiographic evidence of CWP. He acknowledged the miner had at least a moderately severe chronic obstructive pulmonary disease. (Deposition, pg 17). Dr. Castle stated the miner had a moderate degree of airways obstruction in 1989. He noted at this time there was also a significant degree of reversibility consistent with a diagnosis of bronchial asthma. (Deposition, pg 19) Dr. Castle also noted that in 1990 the miner had normal arterial blood gases. (Deposition, pg 21). He agreed the miner would not have had the pulmonary capacity to undertake resection of his tumor. (Deposition, pg 23). However, he disagreed that the surgery would have been "curative." (Deposition, pg 24). Dr. Castle stated the miner had significant COPD due to his long and extensive tobacco habit and that it was not caused by CWP or coal mine dust exposure. (Deposition, pg 25-26). Dr. Castle testified that even if CWP were found to be present, "it was so minimal that…the very minimal abnormality that he would have had I don't believe would have contributed to his death…" (Deposition, pg 29). The remainder of the direct testimony was basically a reiteration of Dr. Castle's conclusions contained within his reports. On cross-examination, Dr. Castle stated he was not in a position to say whether one option, i.e. radiation or surgery, would have been better than the other in treating this type of lung cancer. (Deposition, pg 35-36). He estimated that less than 5% of non-smoking coal miners developed clinically significant COPD. (Deposition, pg 38). He opined the miner had tobacco smoke-induced COPD that represented a significant "asthmatic bronchitic type of process" with some degree of emphysema. Dr. Castle agreed that part of the COPD would be non-asthma related. (Deposition, pg 39). He admitted the miner had emphysema but could not tell what kind of emphysema was present. (Deposition, pg 40). Dr. Castle agreed that dust exposure could contribute to emphysema. (Deposition, pg 41). Dr. Castle agreed that the presence of a coal macule was not necessary in order to have a coal dust relationship to that emphysema. (Deposition, pg 45-46). Dr. Castle stated that he believed the miner could have had centriacinar emphysema since he had no way to rule it out. When asked if the centriacinar emphysema could be related to dust exposure he responded by stating, "It can in a statistically significant way, and I believe that the man does indeed have centriacinar or centrilobular emphysema due to tobacco abuse." (Deposition, pg 46-47). He agreed that the miner had centriacinar emphysema and asthmatic bronchitis and that there was literature that stated this condition could be caused or contributed to by coal dust. Moreover, Dr. Castle could not deny the presence of literature that

supported the assertion that the miner, in this particular case, could have emphysema caused by coal mine dust exposure in addition to that emphysema that was caused by smoking. (Deposition, pg 49). However, Dr. Castle opined, based in part on negative x-rays and CT scans, that the extent of that relationship was very, very, very minimal. (Deposition, pg 49). Further, he stated that this very minimal abnormality, if due to pneumoconiosis, would not have contributed to any clinically significant degree. (Deposition, pg 50). Dr. Castle opined the miner's chronic bronchitis would not be related to coal dust since industrial bronchitis was related to an irritation that was an ongoing process of material being deposited in the larger airways. He added the miner's coal dust exposure ended in 1989 but that another irritation, i.e. smoking, continued. Accordingly, Dr. Castle could not relate the bronchitis to coal mine dust exposure. (Deposition, pg 52). In discussing coal-dust-induced bronchitis, Dr. Castle stated there was "evidence of a statistically significant change but not a clinically significant change." (Deposition, pg 55). Dr. Castle agreed coal mine dust disease could progress after one left the coal mines. (Deposition, pg 56). Dr. Castle admitted it was statistically possible the miner had some minimal degree of emphysema that could have been due to coal mine dust exposure but that it did not cause any clinically significant abnormalities. (Deposition, pg 58).

The supplemental report of Dr. Castle is dated November 4, 2002 and appears at EX 17. Dr. Castle reviewed the medical report of Dr. Cohen and stated that his opinions remained as stated in his previous report and deposition. He added it continued to be his opinion that the miner did not suffer from CWP and that the miner did suffer from tobacco-smoke-induced chronic obstructive pulmonary disease with a significant asthmatic component. Moreover, Dr. Castle added the miner's death was not caused by, contributed to, or hastened in any way by CWP or coal mine dust exposure. It was his opinion the miner would have died as and when he did regardless of his occupational history.

Dr. W.K.C. Morgan

The medical report of Dr. Morgan is dated February 19, 2002 and appears at EX 7. Dr. Morgan is a highly qualified pulmonologist and B-reader of chest x-rays. (EX 7). Dr. Morgan reviewed various x-ray interpretations, medical records from clinics and hospitals, arterial blood gases, pulmonary function studies, and various consultation reports. He concluded the miner died as a result of lung cancer and that he had severe emphysema which was entirely a consequence of cigarette smoking. He added the miner stopped working in 1989 and at that time the miner had mild to moderate airways obstruction as a result of his continued heavy smoking. Dr. Morgan noted the miner was no longer exposed to coal dust after 1989 yet his lung function continued to deteriorate. He opined that since the miner was no longer exposed to coal dust "one would have to" attribute the decline in his ventilatory capacity entirely to his continued habit of cigarette smoking. Dr. Morgan acknowledged that CWP could progress after exposure to coal dust ceases, but for the most part this was related either to the presence of silicosis rather than CWP, or to the development of progressive massive fibrosis in a subject that had definite radiographic evidence of simple CWP. Dr. Morgan concluded this was not the case with the miner. Dr. Morgan opined, after a review of the radiographic evidence, there was insufficient

evidence to justify a diagnosis of CWP. He added the miner had airways obstruction and was grossly impaired but that this was in no way related to CWP, rather it was a consequence of his long continued habit of cigarette smoking for over 50 years. Dr. Morgan stated "there was and is no evidence of CWP and as such it could not play any role in Mr. Williams' death." However, he noted that if histologic evidence of CWP was found, his opinion would not change.

The supplemental report of Dr. Morgan is dated August 27, 2002 and appears at EX 8. Dr. Morgan reviewed additional medical evidence including additional chest x-ray reports and the reports of CT scans. Dr. Morgan concluded, in light of the fact the CT scans taken in 1998 were negative, any diagnosis of CWP should be invalid. He noted that a CT scan was much more sensitive and enabled the lungs to be examined in greater detail. Dr. Morgan stated, "The fact that Drs. Perme, Wiot and Meyer saw no evidence of any CWP invalidates any prior opinion that Mr. Williams has CWP. This does not exclude the presence of an insignificant amount of some soot or coal dust being in Mr. Williams' lungs but *renders a diagnosis of CWP out of the question*." (emphasis added).

The second supplemental report of Dr. Morgan is dated October 2, 2002 and appears at EX 13. Dr. Morgan reviewed the medical report of Dr. Cohen and disagreed with Dr. Cohen's conclusion that CWP was definitely present. Dr. Morgan concluded that as a roof bolter, and using the proper equipment such as a mask, it was "unlikely" the miner would have been exposed to any harmful silica or coal dust. Moreover, he noted the miner had a moderately severe respiratory impairment by the time he developed his lung cancer. Dr. Morgan added that prior to the development of lung cancer there had been a gradual deterioration in the miner's lung function which had developed over the last 15 or so years of the miner's life. He noted that the miner, as a roof bolter, may well have been short of breath on the account of the fact that he had developed COPD. However, Dr. Morgan concluded that coal dust exposure played no role at all in any disability he may have had prior to his death. He added there was no definite evidence of the miner having CWP. Dr. Morgan stated his opinion would not change even if it were found that the miner did have CWP. He also noted that the pathologists who reviewed the various histological slides were of the opinion that there was no evidence of CWP.

Dr. David M. Rosenberg

The medical report of Dr. Rosenberg is dated February 25, 2002 and appears at EX 7. Dr. Rosenberg is Board-Certified in Internal Medicine, Pulmonary Disease, and Occupational Medicine and is a certified B-reader of chest x-rays. (EX 7). Dr. Rosenberg reviewed various medical records from clinics and hospitals, x-ray interpretations, the miner's death certificate, and the medical reports of Drs. Daniel, Rasmussen, Morgan, Naeye, Bush, Caffrey, Zaldivar, and Castle. Dr. Rosenberg noted an occupational history of 23 and ¾ years of coal mine employment. He also noted a smoking history of more than a pack of cigarettes per day from the age of 17. Dr. Rosenberg concluded the miner had severe COPD and that this obstruction was severe enough to disable him from his previous coal mine employment. He acknowledged coal dust exposure could cause the development of COPD and that COPD could occur irrespective of

whether or not a chest x-ray was considered negative. Dr. Rosenberg stated, "The question to be addressed with respect to Mr. Williams, is whether or not severe disabling COPD can occur in an individual absent the complicated form of this illness." After some discussion, Dr. Rosenberg concluded that while coal dust could cause COPD, severe disabling COPD did not occur in relationship to coal mine dust exposure absent the presence of complicated CWP. Accordingly, he opined that the miner's disabling COPD was not the consequence of coal mine dust exposure. In conclusion, Dr. Rosenberg opined the miner did not have CWP. He noted that while the miner had severe disabling COPD, this pulmonary condition and resultant impairment was not hastened or caused by coal mine dust exposure but was etiologically related to cigarette smoking. He added that the miner's death was due to lung cancer and that death was not caused or hastened by the miner's past employment in the coal mines. Further, Dr. Rosenberg noted his opinions would not change even if it were found the miner had a degree of CWP.

The supplemental report of Dr. Rosenberg is dated September 4, 2002 and appears at EX 8. Dr. Rosenberg reviewed excerpts from the ALJ's decision and order in the living miner's claim for benefits, various x-ray interpretations, and the reports of Drs. Wiot, Meyer, and Perme regarding the interpretation of a chest CT scan. Dr. Rosenberg concluded the miner did not have the interstitial form of CWP. He noted that the CAT scan was a much more sensitive instrument for defining the presence or absence of this form of CWP. He noted the miner's CAT scan was negative for CWP, thereby confirming the negative B-readings of the miner's 1990 chest x-ray. Dr. Rosenberg reiterated that the miner's lung cancer and ultimate death were not related to the inhalation of coal mine dust exposure or the development of CWP.

The deposition of Dr. Rosenberg was taken on September 24, 2002 and appears at EX 9. Dr. Rosenberg testified the miner had significant exposure to coal mine dust. He acknowledged the miner had a pulmonary impairment during his lifetime. Dr. Rosenberg noted the radiographic evaluation was consistent with advanced emphysema. He added that in 1989, the pulmonary function data demonstrated the presence of a moderate to severe airflow obstruction. By 1998, the miner had a progression of the obstructive pulmonary impairment. Dr. Rosenberg stated that this obstructive lung disease interfered with gas exchange as far back as 1989. Dr. Rosenberg then acknowledged that CWP could be a progressive lung disease. However, he stated that in this case, in the absence of nodular formation on x-ray, the pattern of progression was not the pattern one would see in progressive massive fibrosis. He attributed the progression in the airways disease to the miner's continued smoking. Dr. Rosenberg acknowledged coal mine dust exposure caused obstructive lung disease. But in this case the severity of the COPD was not characteristic of coal dust exposure "in a setting of his chest x-rays which did not show any micronodular changes, or for that matter, findings of progressive massive fibrosis." Dr. Rosenberg attributed the obstructive lung disease entirely to smoking. The remainder of the direct examination was basically a reiteration of the findings contained within his medical reports. On cross-examination Dr. Rosenberg acknowledged that in his report he stated, "Thus while coal dust exposure can cause COPD…severe disabling COPD does not occur in relationship to coal mine dust exposure

absent complicated CWP." [10] However, Dr. Rosenberg then agreed that simple pneumoconiosis could be disabling. (Deposition, pg 35). Moreover, he added that one could get disabling CWP with any form of simple CWP. (Deposition, pg 36). Dr. Rosenberg testified that the definition of CWP included chronic obstructive pulmonary disease. (Deposition pg 37). He admitted that the miner had a disabling obstructive disease and that the obstructive disease "probably" did contribute to the miner's death. (Deposition, pg 43-44). Dr. Rosenberg testified that although the miner worked in an era where he had the potential to develop a coal mine related pulmonary condition, the miner, to a reasonable degree of probability, was not impacted by the coal mine dust. (Deposition pg 44). Dr. Rosenberg reasoned that the progressive loss of FEV 1 from 1989 to 1998 was over one liter and that this progression occurred after the miner left the coal mines. He also noted that this type of loss was not associated with CWP. (Deposition pg 45). Dr. Rosenberg admitted it was possible, but unlikely, to have disabling CWP absent x-ray findings. (Deposition pg 46). Dr. Rosenberg opined the miner had a "severe form of chronic obstructive pulmonary disease" with a significant emphysematous component and a minimal asthmatic component. (Deposition pg 46-47). Even assuming, hypothetically, that there was a significant reduction in FEV 1 in coal miners from coal mine dust, Dr. Rosenberg opined that he would rule out this coal mine dust exposure as not having "contributed in anything significant in this gentleman." (Deposition pg 55). He based this conclusion on the timing of the loss combined with the "fact that there is no radiographic evidence of any nodule formation" (Deposition pg 56).

The second supplemental report of Dr. Rosenberg is dated October 8, 2002 and appears at EX 12. Dr. Rosenberg reviewed the medical report of Dr. Cohen and agreed that coal dust exposure caused chronic obstructive pulmonary disease. Moreover, he stated that the interstitial form of CWP, not COPD, could progress after miners have been removed from the workplace. He noted that progressive airflow obstruction causing disability after coal mine employment had ceased in the setting of a negative chest x-ray, was not related to past coal dust exposure. Dr. Rosenberg opined the miner could not have had curative surgery due to the presence of Horner's syndrome. He concluded the miner did not have the interstitial form of CWP. He noted that while the miner had disabling COPD, this condition and related disability was a consequence of his long smoking history and not the inhalation of coal mine dust. Dr. Rosenberg stated the lung cancer was smoking-related, and irrespective of the miner's functional status, his lung cancer was not curative in nature. He concluded that CWP did not play any role or did not hasten the miner's death and that his opinion would not change if the miner was found to have a degree of CWP.

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[10] Dr. Rosenberg testified that when he makes statements like the foregoing, he is talking about a medical probability greater than 50 percent. When questioned regarding the phrase "reasonable degree of medical certainty," Dr. Rosenberg responded by stating, "I'm not sure what that means." After further discussion regarding medical probability and medical certainty, Dr. Rosenberg concluded, "It's the same thing probability and medical certainty is the same thing." (Deposition pg 41).

Dr. Robert Andrew Chase Cohen

The medical report of Dr. Cohen is dated September 20, 2002 and appears in the record at CX-1. Dr. Cohen is Board-Certified in Internal Medicine, Pulmonary Disease, and Critical Care Medicine. He is also a B-reader of chest x-rays. Dr. Cohen is currently the Senior Attending Physician of the Pulmonary Division at Cook County Hospital in Chicago. (CX-2). Dr. Cohen reviewed the miner's work history and noted at least 25 years of coal mine employment. He also noted a smoking history of 30 to 45 pack years. He reviewed the miner's past medical history and reviewed the various medical reports of Drs. Daniel, Rasmussen, Zaldivar, Bush, Rosenberg, Morgan, Naeye, Castle, and Caffrey. Dr. Cohen then reviewed x-ray reports, CT Scan reports, and an EKG from 1999. He also reviewed and summarized the pulmonary function studies and arterial blood gases of record. In addition, Dr. Cohen reviewed various treating medical records, outpatient records, and the miner's death certificate. Based on his review of the records, Dr. Cohen concluded the miner had coal workers pneumoconiosis and that his chronic respiratory condition was substantially related to his more than 25 years of coal mine employment and his 30 to 45 pack years of tobacco smoke exposure. Dr. Cohen then set forth in his report the specific basis for that conclusion. Next, Dr. Cohen noted that contrary to the opinions of many of the consultants to this case, there were "numerous findings of modern medical and scientific studies that confirm the link between occupational exposure to coal dust and obstructive lung disease." He went on to discuss various studies that confirm coal dust causes obstructive lung disease with impairment of the FEV-1. Consistent with the opinion of Dr. Morgan but contrary to the opinion of Dr. Castle, Dr. Cohen did not find any convincing evidence that the miner had asthma. However, he noted, even if there was an asthmatic component to the obstructive lung disease, that bronchial hyperactivity would be related to the coal dust exposure. In light of the fact that the miner's last FEV-1 was only 20% of predicted and that he had blood gases showing severe hypoxemia, Dr. Cohen opined that the miner's pneumoconiosis rendered him disabled from his last coal mine employment. In response to the report of Dr. Morgan, Dr. Cohen discussed several studies that supported his position that progression of disease after exposure ceases in no way rules out coal dust as a cause of the miner's impairment. As to the miner's cause of death, Dr. Cohen concluded that the miner clearly died from effects of lung cancer on his already impaired pulmonary function. He noted that when the miner first presented, he had a curable lesion. However, the consulting radiation oncologist stated, "If he didn't have this gross compromised pulmonary function, he would have been a candidate for surgical resection of the tumor as the primary mode of therapy; but with his present condition, this may not be a viable option." Dr. Cohen went on to note that the miner could have had curative surgery had he not had severe chronic obstructive pulmonary disease caused by more than 25 years of exposure to coal mine dust and his 30-45 pack years of tobacco smoke exposure. Dr. Cohen concluded, "The sum of the medical evidence in conjunction with this patient's work history indicates that this patient's more than 25 years of coal mine dust exposure and 30-45 pack year exposure to tobacco smoke was significantly contributory to the development of his severe obstructive lung disease and hypoxemia on blood gases. This disease was significant enough to have caused his total disability for his last coal mining job as a roof bolter helper, and hastened his death from lung cancer."

Conclusions of Law

Length of Coal Mine Employment

The parties have stipulated and I find that the miner was a coal miner within the meaning of the Act for at least 25 years. (TR 13).

Date of Filing

The parties have stipulated and I find that Claimant filed her claim for benefits under the Act on May 25, 2000. (DX-1; TR 13).

Responsible Operator

The parties have stipulated and I find that Consolidation Coal Company is the responsible operator and will provide payment of any benefits awarded to Claimant. (TR 13).

Dependents

At the hearing, Claimant claimed one dependent for purposes of augmentation of benefits under the Act. (TR 11). Counsel noted that Claimant and the miner initiated formal proceedings to adopt their grandson, Steven Alexander prior to the miner's death. The miner died on May 3, 2000 and the adoption order was issued on May 25, 2000. (TR 12; DX 11). The adoption order specified that Steven Alexander was entitled to all of the rights, privileges, and relations existing between a child and its natural parents including the right of inheritance. (DX 11). Claimant testified that Steven lived with them for years before the miner passed away and that she and her husband had been supporting him. (TR 16-17). Claimant testified that Steven was to turn 17 years old that December and that he was attending high school. (TR 17). Claimant also testified she did not receive any child support or payment from Steven's natural parents. (TR 17-18).

At issue is whether a child adopted after the miner's death can be considered a dependent, in a survivor's claim for benefits, for purposes of augmentation of benefits under the Act. The Benefits Review Board addressed this narrow issue in the case of *Blair v. R & E Coal Co.*, 20 BLR 1-16 (1996). In considering this issue, the Board went through the following analysis:

> Section 412(a)(4) of the Act provides that where an individual entitled to benefit payments has one or more dependents, the benefit payments shall be increased at certain stated rates depending upon the number of dependents. 30 U.S.C. § 922(a)(4). Claimant, as the miner's widow, falls within the definition of individuals entitled to receive benefit payments.

30 U.S.C. § 922(a)(2). The relevant inquiry in the instant case is whether claimant's adopted daughter satisfies the definition of a dependent. The definition of a dependent under the Act includes a "child" of the widow who is unmarried and under eighteen years of age. 30 U.S.C. § 902(a)(1), (g).

The Act provides that the determination of an individual's status as the "child" of the widow shall be made in accordance with Section 416(h)(2) or (3) of Title 42 as if the widow were the "insured individual" referred to therein. 30 U.S.C. §902(g). Section 416(h)(2) recognizes an individual as a "child" if that individual could inherit the insured's personal property as his or her natural child under the relevant State inheritance laws. *See* 42 U.S.C. § 416(h)(2)(A); 20 C.F.R. §404.355(a). In the instant case, the adoption decree issued by a Virginia court specifically provides that claimant's adopted child is entitled to all the rights and privileges of a child born in lawful wedlock. (DX 11). Inasmuch as claimant's adopted daughter is eligible to inherit claimant's personal property as her natural child under the Virginia intestacy statutes, the adopted daughter qualifies as claimant's child. Because claimant's adopted child is unmarried and under eighteen years of age, she qualifies as claimant's dependent under the Act. 30 U.S.C. § 902(a)(1), (g).

The regulations also provide for augmented benefits to a miner's widow on behalf of a child adopted after the miner's death. Section 725.520(c)(1) provides that when a surviving spouse is entitled to benefits for a month for which she has one or more dependents, the amount of her benefits is increased. *See* 20 C.F.R. § 725.520(c)(1). However, in order for a surviving spouse to receive augmented benefits on behalf of an individual, that individual is required to satisfy both a relationship and a dependency test. Section 725.208 provides that an individual will be considered to be the child of a beneficiary if the individual is the legally adopted child of the beneficiary. 20 C.F.R. § 725.208(b). Claimant's legally adopted daughter satisfies the relationship test under 20 C.F.R. § 725.208.

In order to receive augmented benefits on behalf of her child, a surviving spouse must also establish that the child is dependent upon her. Section 725.209 provides that a child of a surviving spouse will be determined to be dependent upon such spouse if the child is unmarried and under 18 years of age. *See* 20 C.F.R. § 725.209(a)(1) and (a)(2)(i). Consequently, Claimant's adopted child satisfies this dependency test under 20 C.F.R. § 725.309.

Pursuant to the Board's holding in *Blair, supra*, Claimant, in the instant case, has established that her adopted child, Steven Alexander qualifies as her dependent under the Act and that the adopted child satisfies both the relationship and dependency tests set out in the regulations. (DX 11; TR 16-18). Consequently, if it is found that Claimant is entitled to survivor's benefits, she is entitled to augmented benefits on behalf of her child adopted after the miner's death.

Entitlement to Benefits

Applicable Regulations

Claimant's claim for benefits was filed on May 25, 2000 and is governed by the Part 718 Regulations. However, on January 19, 2001, substantial changes to Parts 725 and 718 of the Federal Regulations became effective. Based upon my review of the new Regulations, there are two sections that specifically deal with the question of whether these new Regulations are applicable to cases that are currently pending at the time of the enactment.

Pursuant to § 725.2(c) the revisions of this part [Part 725] shall also apply to the adjudication of claims that were pending on January 19, 2001, except for the following sections: § 725.309, 725.310, etc. (see the C.F.R. for the complete list of exempted sections). Accordingly, with the exception of those sections listed as an exemption, the revisions to Part 725 will apply to the facts of this decision.

Pursuant to § 718.101(b) the standards for the administration of clinical tests and examinations contained in subpart B "shall apply to all evidence developed by any party after January 19, 2001 in connection with a claim governed by this part [718]..." (emphasis added). Accordingly, since the evidence in the instant matter was developed prior to January 19, 2001, the newly enacted § 718, subpart B does not apply.

On August 9, 2001, U.S. District Court Judge Emmet Sullivan upheld the validity of the new Regulations in *National Mining Association v. Chao*, No. 00-3086 (D.D.C. Aug. 9, 2001). However, on June 14, 2002, the United States Court of Appeals for the District of Columbia Circuit ("the court") affirmed in part, reversed in part, and remanded the case. *See National Mining Association v. Department of Labor*, No. 01-5278 (June 14, 2002). Accordingly, I will apply the sections of the newly revised version of Part 718 (i.e. subparts A, C and D) and 725 that took effect on January 19, 2001 that the court did not find impermissibly retroactive to the facts of the instant matter.

Standard of Review

The administrative law judge need not accept the opinion of any particular medical witness or expert, but must weigh all the evidence and draw his/her own conclusions and inferences. *Lafferty v. Cannelton Industries, Inc.*, 12 BLR 1-190 (1989); *Stark v. Director, OWCP*, 9 BLR 1-36 (1986); *Todd Shipyards Corp. v. Donovan*, 300 F.2d 741 (5th Cir. 1962). The adjudicator's

function is to resolve the conflicts in the medical evidence; those findings will not be disturbed on appeal if supported by substantial evidence. *Lafferty, supra; Fagg v. Amax Coal Co.*, 12 BLR 1-77 (1988), *aff'd*, 865 F.2d 916 (7th Cir. 1989); *Short v. Westmoreland Coal Co.*, 10 BLR 1-127 (1987); *Piccin v. Director, OWCP*, 6 BLR 1-616 (1983). *Peabody Coal Co. v. Lowis*, 708 F.2d 266, 5 BLR 2-84 (7th Cir. 1983).

In considering the medical evidence of record, an administrative law judge must not selectively analyze the evidence. *See Wright v. Director, OWCP*, 7 BLR 1-475 (1984); *Hess v. Clinchfield Coal Co.*, 7 BLR 1-295 (1984); *Crider v. Dean Jones Coal Co.*, 6 BLR 1-606 (1983); *Peabody Coal Co. v. Lowis*, 708 F.2d 266, 5 BLR 2-84 (7th Cir. 1983); *see also Stevenson v. Windsor Power House Coal Co.*, 6 BLR 1-1315 (1984). The weight of the evidence, and determinations concerning credibility of medical experts and witnesses, however, is for the administrative law judge. *Mabe v. Bishop Coal Co.*, 9 BLR 1-67 (1986); *Brown v. Director, OWCP*, 7 BLR 1-730 (1985); *see also Roberts v. Bethlehem Mines Corp.*, 8 BLR 1-211 (1985); *Henning v. Peabody Coal Co.*, 7 BLR 1-753 (1985); *Peabody Coal Co. v. Benefits Review Board*, 560 F.2d 797, 1 BLR 2-133 (7th Cir. 1977).

As the trier-of-fact, the administrative law judge has broad discretion to assess the evidence of record and determine whether a party has met its burden of proof. *Kuchwara v. Director, OWCP*, 7 BLR 1-167 (1984). In considering the evidence on any particular issue, the administrative law judge must be cognizant of which party bears the burden of proof. Claimant has the general burden of establishing entitlement and the initial burden of going forward with the evidence. *See White v. Director, OWCP*, 6 BLR 1-368 (1983)

Entitlement: In General

To establish entitlement to survivor's benefits, claimant must establish that the miner had pneumoconiosis, that the miner's pneumoconiosis arose out of coal mine employment, and that the miner's death was due to pneumoconiosis. 20 C.F.R. §§ 718.3, 718.202, 718.203, 718.205(a); *Trumbo v. Reading Anthracite Co.*, 17 BLR 1-85 (1993); *Haduck v. Director, OWCP*, 14 BLR 1-29 (1990); *Neeley v. Director, OWCP*, 11 BLR 1-85 (1988); *Boyd v. Director, OWCP*, 11 BLR 1-39 (1988). For survivor's claims filed on or after January 1, 1982, the miner's death will be considered due to pneumoconiosis if pneumoconiosis was the cause of the miner's death, was a substantially contributing cause or factor leading to the miner's death, death was caused by complications of pneumoconiosis, or the presumption, relating to complicated pneumoconiosis, set forth at Section 718.304 is applicable. 20 C.F.R. § 718.205(c)(1)-(3). Pneumoconiosis is a substantially contributing cause of death if it hastened the miner's death. 20 C.F.R. § 718.205(c)(5); *Shuff v. Cedar Coal Co.*, 967 F.2d 977, 16 BLR 2-90 (4th Cir. 1992), *cert. denied*, 506 U.S. 1050 (1993).

The Existence of Pneumoconiosis and the Application of Collateral Estoppel

As noted previously, the miner was awarded Black Lung benefits in 1992 by Administrative Law Judge Williams. In his decision, ALJ Williams found that the miner had established the existence of pneumoconiosis by x-ray evidence pursuant to § 718.202(1)(a). This decision was subsequently affirmed by the Benefits Review Board in 1993. The Employer did not appeal the decision of the BRB, accordingly, the award of benefits became final.

However, in the instant survivor claim Employer has decided to contest the issue of the existence of pneumoconiosis even in the absence of autopsy evidence.

This raises the threshold issue of whether the employer is collaterally estopped from re-litigating the existence of coal worker's pneumoconiosis in a survivor's claim where the miner was awarded benefits on a living miner's claim.[11]

For collateral estoppel to apply in the present case, which arises within the jurisdiction of the United States Court of Appeals for the Fourth Circuit, claimant must establish that:

(1) the issue sought to be litigated is identical to the one previously litigated;
(2) the issue was actually determined in the prior proceeding;
(3) the issue was a critical and necessary part of the judgment in the prior proceeding;
(4) the prior judgment is final and valid; and
(5) the party against whom the estoppel is asserted had a full and fair opportunity to litigate the issue in the previous forum.

See Sedlack v. Braswell Services Group, Inc., 134 F.3d 219 (4th Cir. 1998); *Hughes v. Clinchfield Coal Co.*, 21 BLR 1-134 (1999)(*en banc*).

At the time of the adjudication of the miner's claim, evidence sufficient to establish pneumoconiosis under one of the four methods set out at 20 C.F.R. § 718.202(a)(1)-(4) obviated the need to do so under any of the other methods. *See Dixon v. North Camp Coal Co.*, 8 BLR 1-344 (1985). However, subsequent to the issuance of the award of benefits in the miner's claim, the Fourth Circuit held that although Section 718.202(a) enumerates four distinct methods of

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[11] Both parties failed to raise and address the issue of collateral estoppel in their closing briefs. However the courts have generally held that the adjudicator may raise the issue of collateral estoppel *sua sponte*. In *Studio Art Theatre of Evansville, Inc. v. City of Evansville*, 76 F.3d 128, 130 (7th Cir. 1996), the circuit court held that the benefits of precluding re-litigation of issues runs not only to the litigants, but to the judicial system. Moreover, in *Doe v. Pfrommer*, 148 F.3d 73 (2d Cir. 1998), it was held that "strong public policy in economizing the use of judicial resources by avoiding re-litigation "favors *sua sponte* application of collateral estoppel." *See also Tri-Med Finance Co. v. National Century Finance Enterprises, Inc.*, 208 F.3d 215 (6th Cir. 2000).

establishing pneumoconiosis, all types of relevant evidence must be weighed together to determine whether a miner suffers from the disease. *See Island Creek Coal Co. v. Compton*, 211 F.3d 203, ___ BLR ___ (4th Cir. 2000); *see also Penn Allegheny Coal Co. v. Williams*, 114 F.3d 22, 21 BLR 2-104 (3d Cir. 1997). In light of the change in law enunciated in *Compton*, which overruled the Board's holding in *Dixon*, the issue of whether the existence of pneumoconiosis has been established pursuant to Section 718.202(a) is not identical to the one previously litigated and actually determined in the miner's claim. *See Sedlack, supra*; *Hughes, supra*. Thus, inasmuch as each of the prerequisites for application of the doctrine of collateral estoppel is not present, I find that the doctrine of collateral estoppel is not applicable in this survivor's claim regarding the existence of pneumoconiosis pursuant to 20 C.F.R. § 718.202(a). *See Surway v. United Pocahontas Coal Co*, BRB No. 01-0881 BLA (June 26, 2002)(unpub.); *Howard v. Valley Camp Coal Co.*, BRB No. 001034 BLA (August 24, 2001)(unpub.); *Price v. Consolidated Coal Co.*, BRB No. 00-0453 BLA (January 24, 2001)(unpub.). Consequently, I will reconsider the evidence and determine whether it is sufficient to establish the existence of pneumoconiosis in accordance with the standard enunciated in *Compton.*[12]

[12] *The instant case is distinguishable from the recent case of Zeigler Coal Co. v. Director, OWCP [Villain]*, ___F.3d___, Case No. 01-3961 (7th Cir. December 6, 2002). In that case the Seventh Circuit held that an employer is collaterally estopped from re-litigating the existence of pneumoconiosis in a survivor's claim where the miner was awarded benefits based on a lifetime claim and no autopsy evidence is presented in the survivor's claim. In that case, the claimant only had to prove the existence of pneumoconiosis within one of the discrete subsections of § 718.202(a). After the underlying case in *Zeigler* was decided (on December 7, 1999), the Fourth Circuit adopted the new standard enunciated in *Compton, supra*. The Seventh Circuit has not yet ruled on this issue of weighing evidence together under § 718.202(a)(1)-(4). Therefore, since there has not been any change in the law in the Seventh Circuit regarding the weighing of the evidence under this subsection, the issue in the survivor's claim was identical to the issue in the living miner's claim and collateral estoppel could be applied. Conversely, in the instant case, pursuant to the Fourth Circuit's recent holding in *Compton, supra*, the issue now presented in the survivor's claim is not identical to the issue that was presented in the living miner's claim in 1992. Therefore, since all of the prerequisites for the application of collateral estoppel are not present, the doctrine of collateral estoppel is not applicable in the instant survivor's claim regarding the existence of pneumoconiosis.

The Existence of Pneumoconiosis

Thirty U.S.C. § 902(b) and 20 C.F.R. § 718.201 defines pneumoconiosis as a "a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment." [13] The definition is not confined to "coal workers' pneumoconiosis," but also includes other diseases arising out of coal mine employment, such as anthracosilicosis, anthracosis, anthrosilicosis, massive pulmonary fibrosis, progressive massive fibrosis, silicosis, or silicotuberculosis. 20 C.F.R. § 718.201. The term "arising out of coal mine employment" is defined as including "any chronic pulmonary disease resulting in respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment."

" . . . [T]his broad definition 'effectively allows for the compensation of miners suffering from avariety of respiratory problems that may bear a relationship to their employment in the coal mines.'" *Robinson v. Pickands Mather & Co./Leslie Coal Co. & Director, OWCP*, 14 B.L.R. 2-68 (4th Cir. 1990) at 2-78, 914 F.2d 35 (4th Cir. 1990) *citing*, *Rose v. Clinchfield Coal Co.*, 614 F. 2d 936, 938 (4th Cir. 1980). Thus, asthma, asthmatic bronchitis, or emphysema may fall under the regulatory definition of pneumoconiosis if they are related to coal dust exposure. *Robinson v. Director, OWCP*, 3 B.L.R. 1-798.7 (1981); *Tokarcik v. Consolidation Coal Co.*, 6 B.L.R. 1-666 (1983). Likewise, chronic obstructive pulmonary disease may be encompassed within the legal definition of pneumoconiosis. *Warth v. Southern Ohio Coal Co.*, 60 F.3d 173 (4th Cir. 1995).

The claimant has the burden of proving the existence of pneumoconiosis by any one of four methods. The Regulations provide the means of establishing the existence of pneumoconiosis by: (1) a chest X-ray meeting the criteria set forth in 20 C.F.R. § 718.202(a); (2) a biopsy or autopsy conducted and reported in compliance with 20 C.F.R. § 718.106; (3) application of the irrefutable presumption for "complicated pneumoconiosis" found in 20 C.F.R. § 718.304; or (4) a determination of the existence of pneumoconiosis made by a physician exercising sound judgment, based upon certain clinical data and medical and work histories, and supported by a reasoned medical opinion. 20 C.F.R. § 718.202(a). Pulmonary function studies are not diagnostic of the presence or absence of pneumoconiosis. *Burke v. Director, OWCP*, 3 B.L.R. 1-410 (1981).

In *Island Creek Coal Co. v. Compton*, 211 F.3d 203, 2000 WL 524798 (4th Cir. 2000), the Fourth Circuit held that the administrative law judge must weigh all evidence together under 20 C.F.R. § 718.202(a) to determine whether the miner suffered from coal workers' pneumoconiosis. This is contrary to the Board's view that an administrative law judge may weigh the evidence under each subsection separately, *i.e.* x-ray evidence at § 718.202(a)(1) is weighed apart from the medical opinion evidence at § 718.202(a)(4). In so holding, the court cited to the Third Circuit's

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[13] *Pneumoconiosis is a progressive and irreversible disease; once present, it does not go away. Mullins Coal Co. v. Director, OWCP,* 484 U.S. 135, 151 (1987); *Lisa Lee Mines v. Director,* 86 F.3d 1358 (4th Cir. 1996)(*en banc*) at 1364; *LaBelle Processing Co. v. Swarrow,* 72 F.3d 308 (3d Cir. 1995) at 314-315.

decision in *Penn Allegheny Coal Co. v. Williams*, 114 F.3d 22, 24-25 (3d Cir. 1997) which requires the same analysis.

Chest X-ray Evidence

A finding of the existence of pneumoconiosis may be made with positive chest x-ray evidence. 20 C.F.R. § 718.202(a)(1). The existence of pneumoconiosis may be established by chest x-rays classified as category 1, 2, 3, A, B, or C according to ILO-U/C International Classification of Radiographs. A chest x-ray classified as category 0, including subcategories 0/-0/0, 0/1, does not constitute evidence of pneumoconiosis. 20 C.F.R. § 718.102(b). Where two or more x-ray reports are in conflict, the radiologic qualifications of the physicians interpreting the x-rays must be considered. § 718.201(a)(1).

While a judge is not required to defer to the numerical superiority of x-ray evidence, it is within his or her discretion to do so. *Wilt v. Woverine Mining Co.*, 14 B.L.R. 1-70 (1990) *citing Edmiston v. F & R Coal*, 14 B.L.R. 1-65 (1990). The ALJ must rely on the evidence which he deems to be most probative, even where it is contrary to the numerical majority. *Tokarcik v. Consolidation Coal Co.*, 6 B.L.R. 1-666 (1984).

In addition, the Fourth Circuit noted that pneumoconiosis is "progressive and irreversible" such that it is proper to accord greater weight to later positive x-ray studies over earlier negative studies. It stated further that generally, "later evidence is more likely to show the miner's current condition" where it is consistent in demonstrating a worsening of the miner's condition. *Lane Hollow Coal Co. v. Director, OWCP [Lockhart]*, 137 F.3d 799 (4th Cir. 1998).

Of the submitted evidence, there are sixty-three (63) interpretations of twelve (12) x-rays and one (1) CT Scan in the record. First, I accord less weight to the B-reading of Dr. Rosenberg of the March 25, 1998 x-ray (EX 8) because the x-ray was deemed unreadable by Dr. Rosenberg. *Gober v. Reading Anthracite Co.*, 12 BLR 1-67 (1988). Next, I accord less weight to the reading of Dr. Deghan of the March 15, 1998 x-ray (DX 15) and the readings of Dr. Patel of the March 21, 1998, March 22, 1998, March 23, 1998, and March 25, 1998 chest x-rays (DX 15). These x-rays were taken in the hospital for purposes of diagnosing cancer and/or monitoring a right pneumothorax and not for purposes of diagnosing the presence of pneumoconiosis. I find that given the setting in which these x-rays were taken and read that the omission of a finding of pneumoconiosis in this instance does not necessarily mean that it was not present. For this reason I accord these readings less weight. *See Sacolick v. Rushton Mining Co.*, 6 B.L.R. 1-930 (1984).

Of the remaining fifty-seven (57) interpretations of seven (7) x-rays, fifty-one (51) were negative (a profusion of 0/1 or 0/0 in the ILO classification) and six (6) were positive for pneumoconiosis (a profusion of 1/0 or higher in the ILO Classification). I find that prior to the most recent x-ray of December 26, 1990, the vast majority of the x-ray evidence was negative for pneumoconiosis. However, of the six (6) interpretations of the December 26, 1990 x-ray, four (4) were positive for pneumoconiosis and two (2) were negative for pneumoconiosis. The four

positive interpretations were by B-readers while the two negative interpretations were by dually qualified Board-Certified Radiologists and B-readers. The Board has held that it is proper to credit the interpretation of a dually qualified physician over the interpretation of a B-reader. *Cranor v. Peabody Coal Co.*, 22 BLR 1-1 (1999). (en banc on recon.). However, an administrative law judge may utilize any reasonable method of weighing such evidence. *See Sexton v. Director, OWCP*, 725 F.2d 213 (6th Cir. 1985). Even accounting for the fact that the two negative readings were by dually qualified physicians, I still find that the four highly probative positive B-readings outweigh the two negative readings. Moreover, because the final x-ray was taken almost one year later than the previous negative x-ray, I find that it is more probative of the miner's respiratory condition at the time of death. *See Lockhart, supra*; *Clark v. Karst-Robbins Coal Co.*, 12 BLR 1-149 (1989).[14]

Additionally, Employer submitted the interpretations of Drs. Wiot, Meyer, and Perme of a CT scan taken on March 18, 1998. (DX 18; EX 5). All three are Board-Certified Radiologists and B-readers. All three physicians found no evidence of pneumoconiosis. Moreover, Employer had several of their consultants submit supplemental reports advocating the position that a CT scan was more sensitive and more accurate for determination of parenchymal changes than a standard x-ray. (EX 8). However, in *Consolidation Coal Co. v. Director, OWCP [Stein]*, 294 F.3d 885 (7th Cir. 2002), the court disagreed with Employer's position and noted that the Department has rejected the view that a CT-scan, by itself, "is sufficiently reliable that the negative result effectively rules out the existence of pneumoconiosis." 65 Fed. Reg. 79, 920, 79, 945 (Dec. 20, 2000). Moreover, in the instant matter, there was evidence that the CT scan was a conventional (slices taken at 10 mm intervals) as opposed to a high resolution CT scan (slices taken at 1 mm intervals) and that it was taken for the purposes of diagnosing cancer. (EX 8, 8-22-02 medical report of Dr. Zaldivar). As Dr. Zaldivar pointed out, the benefit of a chest x-ray is that it shows the entire lung at one time rather than small slices through 10 mm cuts throughout the lung. Accordingly, although all three interpretations were read by dually qualified physicians, I find that the CT scan evidence is less probative and credible than the positive x-ray evidence.

Therefore, I find that Claimant has established, by the preponderance of the evidence, the existence of pneumoconiosis pursuant to § 718.202(a)(1).

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[14] Judge Williams reached a similar result in his Decision and Order - Awarding Benefits. (DX 27-31). Moreover, this finding, i.e. the existence of pneumoconiosis pursuant to § 718.202(a)(1), was upheld on appeal by the Benefits Review Board. (DX 27-54).

Biopsy Evidence

A biopsy may be the basis for a finding of the existence of pneumoconiosis. § 718.202(a)(2). A finding in a biopsy of anthracotic pigmentation, however, shall not be sufficient, by itself, to establish the existence of pneumoconiosis. § 718.202(a)(2).[15]

In the instant matter there were three sets of histologic slides containing tissue taken from right lung biopsies. The slides were identified as follows: one slide labeled S98-01541, one slide labeled S98-01393, and five slides labeled C99-4017. Drs. Bush, Naeye, and Caffrey reviewed the slides microscopically and found that the tissue samples contained small to moderate amounts of fine black dust. Drs. Bush and Caffrey concluded that the biopsy tissue was too limited with an inadequate amount of lung tissue to either confirm or deny the presence of CWP. On the other hand, Dr. Naeye never directly stated that the samples were inadequate for evaluation but concluded that the "*tissue provided"* did not have clearly identifiable anthracotic macules or larger black deposits around which focal emphysema could be identified. Based on what Dr. Naeye observed he concluded that the "*available tissue findings*" did not support the diagnosis of CWP.[16]

I accord less weight to the opinion of Dr. Naeye because of his failure to give an assessment regarding the quality and scope of the tissue samples in making his evaluation. Conversely, I accord greater weight to the opinions of Drs. Bush and Caffrey who noted the tissue samples were inadequate to either confirm or deny the presence of pneumoconiosis.

In conclusion, I find that although there was some indication of the presence of black pigment in the biopsy tissues, the samples were inadequate to make a definitive diagnosis. Accordingly, I find that Claimant has failed to establish the presence of pneumoconiosis, by the preponderance of the evidence, pursuant to § 718.202(a)(2).

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[15] *However, in the recent case of Taylor v. Director, OWCP*, BRB No. 01-0837 BLA (July 30, 2002) (unpubl.), the Board noted that while a diagnosis of anthracotic pigmentation is insufficient by itself to establish the existence of pneumoconiosis, both the former and the amended versions of 20 C.F.R. § 718.201 identify "anthracosis" as a disease within the definition of "pneumoconiosis." The Board further held that anthracosis found in lymph nodes may be sufficient to establish the existence of pneumoconiosis. *See Hapney v. Peabody Coal Co.*, 22 BLR 1-104 (2001)(*en banc*).

[16] *Perhaps one explanation why Dr. Naeye could not identify these characteristics of CWP was because the tissue samples, themselves, were inadequate, as Drs. Bush and Caffrey concluded.*

The Presumptions

If the presumptions described in §§ 718.304, 718.305 or 718.306 are applicable, it shall be presumed that the miner is or was suffering from pneumoconiosis. § 718.202(a)(3). I find that none of the foregoing presumptions are applicable in this matter. Therefore, I find that Claimant has failed to establish the presence of pneumoconiosis pursuant to § 718.202(a)(3).

Medical Opinion Evidence

Additionally, a determination of the existence of pneumoconiosis can be made if a physician, exercising sound medical judgment, based upon certain clinical data and medical and work histories and supported by a reasoned medical opinion, finds the miner suffers or suffered from pneumoconiosis, as defined in § 718.201, notwithstanding a negative x-ray. 20 C.F.R. § 718.202(a). Medical reports which are based upon and supported by patient histories, a review of symptoms, and a physical examination constitute adequately documented medical opinions as contemplated by the Regulations. *Justice v. Director, OWCP*, 6 B.L.R. 1-1127 (1984). However, where the physician's report, although documented, fails to explain how the documentation supports its conclusions, an Administrative Law Judge may find the report is not a reasoned medical opinion. *Smith v. Eastern Coal Co.*, 6 B.L.R. 1-1130 (1984). A medical opinion shall not be considered sufficiently reasoned if the underlying objective medical data contraindicates it. *White v. Director, OWCP*, 6 B.L.R. 1-368 (1983).

In the instant matter, eleven physicians submitted reports regarding the miner's medical condition. In general, Drs. Bush, Naeye, Caffrey, Daniel, Zaldivar, Morgan, Castle, and Rosenberg found no evidence of pneumoconiosis. While Drs. Rasmussen, Cohen, and Gaziano concluded that the miner had coal workers pneumoconiosis.

In general, more weight may be accorded to the conclusions of a treating physician as he is more likely to be familiar with the miner's condition than a physician who examines him episodically. *Onderko v. Director, OWCP*, 14 BLR 1-2 (1989). Dr. Rasmussen was the only treating physician to render an opinion in this matter and therefore, his opinion, if well-reasoned and well-documented, may be entitled to greater weight. *McClendon v. Drummond Coal Co.*, 12 BLR 2-108 (11th Cir. 1988).

Section 718.104(d) codifies the "treating physician rule" and provides the following list of factors in weighing the opinion of the miner's treating physician: (1) nature of the relationship, (2) duration of the relationship, (3) frequency of the treatment, and (4) extent of treatment. Based on the medical records, Dr. Rasmussen treated the miner from September of 1989 through March of 2000. The miner was initially referred to Dr. Rasmussen for breathing problems. Dr. Rasmussen treated and evaluated the miner approximately every two to three months (sometimes longer intervals) for more than ten years. Dr. Rasmussen, who was a physician in the Division of Pulmonary Medicine at the Southern West Virginia Clinic, treated the miner primarily for his pulmonary condition. (DX 14). Based on the foregoing, I find that Dr. Rasmussen, as the miner's treating physician, was in a unique position to render an opinion in this matter. Accordingly, if I

find that his opinion is well-reasoned and well-documented, Dr. Rasmussen's opinion should be accorded more weight.

I accord greater weight to the opinions of Drs. Rasmussen and Cohen. I find that Dr. Rasmussen's reports[17] of March 14, 1991 and August 4, 1992 (DX 14) and Dr. Cohen's report of September 20, 2002 (CX 1) are well-documented. *Fields v. Island Creek Coal Co.*, 10 BLR 1-19 (1987). In his reports, Dr. Rasmussen clearly sets forth his clinical findings and observations upon which he based his diagnosis of coal workers' pneumoconiosis. Likewise, Dr. Cohen clearly sets forth, in a point-by-point manner, his reasons for concluding the miner had coal worker's pneumoconiosis. Moreover, Drs. Rasmussen and Cohen properly took into consideration the miner's significant history of approximately 24 years of underground coal mine employment and his substantial history of heavy cigarette smoking.[18] *Hoffman v. B&G Construction Co.*, 8 BLR 1-65 (1985). I also find that the opinions of Drs. Rasmussen and Cohen are well-reasoned. *Fields, supra*. I find that the underlying documentation contained within their reports is adequate to support the conclusions of Drs. Rasmussen and Cohen that the miner had coal worker's pneumoconiosis. Dr. Rasmussen noted the disparity in the interpretation of the chest x-rays and noted that pneumoconiosis could be present in significant degree without being visible by x-ray (clinical pneumoconiosis). Moreover, Dr. Rasmussen noted the miner had, in 1989, a moderate, partially reversible obstructive ventilatory impairment. He added that there were two risk factors for the respiratory insufficiency: the miner's history of underground coal mine employment (legal pneumoconiosis) and his significant smoking history. After noting that both risk factors caused similar physiological damage to the lungs, Dr. Rasmussen reasonably concluded that it was impossible to separate the effects of smoking from coal mine dust exposure. Likewise, Dr. Cohen does not try to minimize the miner's significant, heavy smoking history as a contributing factor in his respiratory ailments but does indicate that coal mine dust exposure does cause many of the same findings as smoking (i.e. obstructive defect) and that it was not possible to exclude coal mine dust exposure as at least one factor in the miner's development of severe COPD (legal pneumoconiosis). For these reasons, I accord more weight to the opinions of Drs. Rasmussen and Cohen.

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[17] Although the 2-6-90 medical report of Dr. Rasmussen is missing from the case file, I find Dr. Rasmussen's subsequent report of 4-14-91 to be comprehensive, thorough and based on the totality of the evidence at his disposal, including his 2-6-90 report. (DX 14). Accordingly, I find that the contents of the 2-6-90 report of Dr. Rasmussen would merely be cumulative and its absence from the file is inconsequential and would have no effect on the weighing of Dr. Rasmussen's opinion on this issue.

[18] The miner testified at his hearing in 1991 that he smoked one pack of cigarettes per day for 30 years. (DX 27-30, page 27). Judge Williams accepted this testimony and found the miner had a smoking history of 30 pack years and continuing. (DX 27-31). There is some indication in the medical records that the miner may have smoked up to 1 ¼ packs per day. Dr. Rasmussen noted that in 1996 the miner stopped smoking. (DX 14; March 4, 1997 medical office note). There is no evidence to the contrary in the record. The miner died in 2000 at the age of 70. (DX 13). Accordingly, I find that the miner had a smoking history of approximately 35 to 44 pack years.

Moreover, due to the fact that I found Dr. Rasmussen's reports to be well-reasoned and well-documented, I find that as the miner's treating physician, his opinion should be accorded greater weight pursuant to § 718.104(d).

I accord less weight to the opinion of Dr. Gaziano on this issue. Pursuant to his brief report, Dr. Gaziano based his diagnosis of pneumoconiosis, at least in part, on the biopsy findings that showed the existence of black pigment. As noted earlier in this opinion, I accorded the biopsy findings little probative value as the weight of the better reasoned opinions suggested that the histologic samples contained inadequate tissue for a proper review and were deemed unreliable. Because Dr. Gaziano based his diagnosis, at least in part, on these biopsy findings, I find that his opinion is not well-documented and is entitled to less weight.

I accord less weight to the opinions of Drs. Bush, Naeye, and Caffrey. Although he is a highly qualified pathologist, I find the opinion of Dr. Bush, regarding the existence of pneumoconiosis, to be equivocal. *Island Creek Coal Co. v. Holdman*, 202 F.3d 873 (6th Cir. 2000). Dr. Bush indicated in one report that there was no "significant" degree of CWP based on a CT scan and x-rays. (DX 17). He noted in another report that "significant" CWP sufficient to cause impairment or death was not present. (EX 8). Once again, Dr. Bush stated in his last report that there was no objective evidence coal mine dust exposure played a "significant" role in the events leading to the miner's death. (EX 15). Although Dr. Bush never definitively diagnoses the presence of pneumoconiosis, I find that these statements and the repeated use of the word "significant" indicate the possibility that pneumoconiosis may be present in some degree. I find these statements regarding the possible existence of pneumoconiosis to be equivocal and at best unclear. Therefore, I accord less weight to the opinion of Dr. Bush.

Likewise, although he is a highly qualified pathologist, I find the opinion of Dr. Naeye to be equivocal as to whether some form of pneumoconiosis was actually present in the miner. Like Dr. Bush, Dr. Naeye never definitively diagnoses the presence of pneumoconiosis. However, Dr. Naeye makes several statements that indicate the possible presence of pneumoconiosis. Dr. Naeye noted in a report that most of the x-rays were negative for pneumoconiosis but added that this fact did not exclude the possibility of its presence in a mild form (i.e. possible clinical pneumoconiosis). (DX 19). Dr. Naeye then acknowledged the presence of a moderately severe obstructive impairment. He noted that had the miner been a non-smoker, it would be "unlikely" that the miner would have developed this airways obstruction thereby suggesting that coal mine dust exposure played no role in the miner's obstructive impairment (i.e. no legal pneumoconiosis). (DX 19). However, in response to the report of Dr. Cohen who concluded that the miner's pulmonary insufficiency and death were in large part due to the result of occupational exposures to coal mine dust, Dr. Naeye stated that the effects of smoking were x4.5 greater than the effects of coal mine dust exposure. Dr. Naeye attempted to minimize the effects of coal mine dust exposure in comparison to smoking, but did not make any convincing argument to rule out coal mine dust exposure as a possible contributing factor *of some kind* to the miner's pulmonary condition (i.e. possible legal pneumoconiosis). (EX 11). Moreover, in another report, he noted that the "available tissue findings" did not support the diagnosis of pneumoconiosis (i.e. no clinical pneumoconiosis). Dr. Naeye then stated that CWP lesions were "not routinely visible" in

images of the miner's lungs but then noted that this did not mean that they were completely absent from his lungs (i.e. possible clinical pneumoconiosis). (EX 6). Because of the equivocal nature of his opinion on this issue, I accord the opinion of Dr. Naeye less weight.

Likewise, although he is a highly qualified pathologist, I find the opinion of Dr. Caffrey to be equivocal on this issue. After reviewing tissue samples from the biopsy, Dr. Caffrey concluded he could not make a diagnosis of pneumoconiosis but also that he could not rule it out. (EX 1). In one of his reports, he assumed that if the miner had pneumoconiosis it was too mild by itself to cause pulmonary disability. (EX 6). In another report, after noting that there was no evidence that the miner "definitely" had pneumoconiosis, Dr. Caffrey stated that miner "may have had" simple coal worker's pneumoconiosis but that a diagnosis was not "absolutely" established. (EX 14). Because of the equivocal nature of Dr. Caffrey's opinion regarding the existence of pneumoconiosis, I accord his opinion less weight.

I accord less weight to the opinion of Dr. Daniel who found no pneumoconiosis to be present. In his report, after acknowledging a negative chest x-ray (i.e. no clinical pneumoconiosis), Dr. Daniel diagnosed the miner as having COPD due to 30 years of smoking based on history of productive cough, history of smoking, and evidence of an obstructive defect (i.e. no legal pneumoconiosis). I find that Dr. Daniel's opinion is not well-reasoned inasmuch as he failed to explain how he was able to completely eliminate the miner's significant coal mine dust exposure as a possible contributing factor to the noted obstructive defect. *Clark v. Karst-Robbins Coal Co.*, 12 BLR 1-149 (1989)(en banc). For this reason, I accord the opinion of Dr. Daniel less weight on this issue.

Although Dr. Morgan is a highly qualified pulmonologist, I accord less weight to his opinion. In one of his reports, Dr. Morgan stated it was unlikely the miner was exposed to harmful coal dust or silica in his 24 years of underground coal mine employment. (EX 13). I find that this statement is pure supposition and has no basis in fact. Moreover, I find this statement is at odds with the opinion of every other physician in the record who directly or impliedly agreed that the miner had sufficient exposure to coal mine dust to be a potential susceptible host of pneumoconiosis. Because the fundamental foundation of Dr. Morgan's opinion is seriously flawed, I accord less weight to his opinion. *Goss v. Eastern Assoc. Coal Co.*, 7 BLR 1-400 (1984).

Although Dr. Rosenberg is a highly qualified pulmonologist, I accord less weight to his opinion. Dr. Rosenberg concluded the miner did not have clinical pneumoconiosis based on the x-ray evidence and CT scan. He noted that the question to be addressed was whether or not severe disabling COPD could occur in an individual absent the complicated form of this illness. He acknowledged coal dust exposure could cause the development of COPD even if a chest x-ray was negative. (EX 7). He then concluded that while coal mine dust could cause COPD, severe disabling COPD did not occur in relationship to coal mine dust exposure absent the presence of complicated CWP. Based on the forgoing reasoning, Dr. Rosenberg opined that the miner's disabling COPD was not the consequence of coal mine dust exposure (i.e. no legal pneumoconiosis). (EX 7). I find this all or nothing approach by Dr. Rosenberg to be less credible

and less persuasive than the well-reasoned reports of Drs. Cohen and Rasmussen. It is well settled that coal mine dust does not have to be the *sole* cause of the COPD in order for there to be a diagnosis of legal pneumoconiosis. The definition of legal pneumoconiosis includes any chronic lung disease arising out of coal mine employment. § 718.201(a)(2). A disease "arising out of coal mine employment" includes any chronic pulmonary disease significantly related to, or substantially aggravated by dust exposure in the coal mine. § 718.201(a)(3). Therefore, it is inappropriate for Dr. Rosenberg to use the absence of complicated pneumoconiosis as the sole criterion to exclude coal mine dust as a possible factor in causing the miner's severe COPD. Based on the foregoing, I find the opinion of Dr. Rosenberg is not well-reasoned and should be accord less weight.

I find the opinion of Dr. Castle, who is also highly qualified, to be equivocal regarding the presence of pneumoconiosis.[19] In his initial report, Dr. Castle stated that even in the absence of radiographic evidence of pneumoconiosis, one could still have pathologic evidence of the disease, thereby suggesting the possibility that clinical pneumoconiosis may exist in this case. (EX 7). Dr. Castle diagnosed the presence of emphysema and asthma, both due to smoking (i.e. no legal pneumoconiosis). (EX 7). However, at his deposition, Dr. Castle admitted that dust exposure could contribute to the development of emphysema and then later agreed that it was possible that a minimal degree of emphysema was due to coal mine dust exposure (i.e. legal pneumoconiosis).[20] (EX 10). Because of the equivocal nature of Dr. Castle's opinion regarding the existence of pneumoconiosis, I accord his opinion less weight on this issue.

Finally, I accord less weight to the highly qualified opinion of Dr. Zaldivar.[21] Dr. Zaldivar diagnosed the miner as having asthma due to a genetic predisposition and emphysema due to smoking. (EX 2). He maintained that the miner did not have coal worker's pneumoconiosis. I find that Dr. Zaldivar's reports are not as well-reasoned and well-documented as the reports of Drs. Cohen and Rasmussen. It is well settled that coal dust exposure can be related to the development asthma and emphysema. *See Robinson v. Director, OWCP*, 3 BLR 1-798.1 (1981).

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[19] Although the April 18, 1991 medical report of Dr. Castle is missing from the case file, I find Dr. Castle's subsequent reports to be comprehensive, thorough and based on the totality of the evidence at his disposal. Accordingly, I find that the contents of the April 18, 1991 report of Dr. Castle would merely be cumulative and its absence from the file is inconsequential and would have no effect on the weighing of Dr. Castle's opinion on this issue.

[20] *See Robinson v. Director, OWCP*, 3 BLR 1-798.7 (1981) (emphysema related to coal dust exposure falls under the regulatory definition of pneumoconiosis)

[21] Although the April 23, 1991 deposition testimony of Dr. Zaldivar is missing from the case file, I find Dr. Zaldivar's February 26, 1990 report, on which the deposition was based, to be comprehensive, thorough and based on the totality of the evidence at his disposal. Accordingly, I find that the deposition testimony would have been basically a reiteration of the contents of the February 26, 1990 report and its absence from the file is inconsequential. Moreover, Dr. Zaldivar subsequently submitted comprehensive reports that contained discussion of all of the relevant evidence at his disposal.

However, Dr. Zaldivar does not adequately explain how he was able to rule out coal dust exposure as a factor in the formation of either disease. For this reason, I find that his opinion is not well-reasoned and as such should be accorded less weight.

In summary, based on the conclusions of the better reasoned opinions, I find that Claimant has established the existence of pneumoconiosis pursuant to § 718.202(a)(4).

Weighing all Evidence Together

Pursuant to the holding in *Compton, supra* I must weigh all of the evidence under § 718.202(a) together in order to make a determination regarding the existence of pneumoconiosis. I found previously that Claimant was able to establish the existence of pneumoconiosis through x-ray evidence pursuant to § 718.202(a)(1). I found that the biopsy evidence was insufficient to establish the existence of pneumoconiosis pursuant to § 718.202(a)(2) and that the presumptions at § 718.202(a)(3) were inapplicable to the facts of the instant matter. In addition, I found that the conclusions of the better reasoned opinions established the existence of pneumoconiosis pursuant to § 718.202(a)(4). Accordingly, weighing all of the foregoing evidence together, I find that Claimant has established the existence of pneumoconiosis pursuant to § 718.202(a).

Pneumoconiosis Arose Out of Coal Mine Employment

Pursuant to §718.203(b) if a miner who is suffering or suffered from pneumoconiosis was employed for ten years or more in one or more coal mines, there shall be a rebuttable presumption that the pneumoconiosis arose out of such employment. Employer did not submit any evidence to rebut said presumption. Accordingly, as the miner worked for more than ten years in a coal mine, I find that Claimant has established that the pneumoconiosis arose out of the miner's coal mine employment pursuant to § 718.203.

Death Due to Pneumoconiosis

The remaining issue is whether the miner's death was due to pneumoconiosis.

Subsection 718.205(c) applies to survivor's claims filed on or after January 1, 1982 and provides that death will be due to pneumoconiosis if any of the following criteria are met:

(1) competent medical evidence established that the miner's death was due to pneumoconiosis; or
(2) pneumoconiosis was a substantially contributing cause or factor leading to the miner's death or the death was caused by complications of pneumoconiosis; or
(3) the presumption of § 718.304 [complicated pneumoconiosis] is applicable.

Pursuant to § 718.205(c)(5), pneumoconiosis is a substantially contributing case of a miner's death if it hastens the miner's death.

There is no evidence that pneumoconiosis was the direct cause of the miner's death, therefore Claimant has not proven death due to pneumoconiosis pursuant to § 718.205(c)(1). There is no evidence the miner suffered from complicated pneumoconiosis, therefore Claimant has not established death due to pneumoconiosis pursuant to § 718.205(c)(3).

There are nine physicians who have rendered an opinion regarding the cause of death. Drs. Gaziano and Cohen found that coal worker's pneumoconiosis was a contributing, or hastening, factor in the miner's death. Drs. Bush, Naeye, Caffrey, Zaldivar, Castle, Morgan, and Rosenberg opined that coal mine dust played no role in hastening the miner's death.

I accord great weight to the highly qualified opinion of Dr. Cohen on this issue. His opinion is well-reasoned and well-documented. *Fields, supra.* Dr. Cohen reasonably concluded, based on the totality of the evidence, that the miner's coal worker's pneumoconiosis and chronic respiratory condition was substantially related to 25 years of coal mine employment and the miner's heavy smoking history. He added that the miner died from the effects of lung cancer on an already impaired pulmonary function. Moreover, he added that the miner had a curable lesion but was prevented from having surgery due to his severe pulmonary dysfunction. This finding is supported by the opinion of Dr. Gaziano who also noted that curative surgery was thwarted by severe lung disease due to coal worker's pneumoconiosis and emphysema. (DX 16). Dr. Cohen concluded that the miner's occupational history and smoking history was significantly contributory to the development of severe obstructive lung disease and hypoxemia on blood gases. He added that this disease was significant enough to have caused total disability from his last coal mining job and hastened the miner's death from lung cancer. Accordingly, I find the medical opinion of Dr. Cohen to be credible and persuasive and as such will be accorded greater weight.

Although Dr. Rosenberg is a highly qualified pulmonologist, I accord less weight to his opinion because of certain inconsistencies in statements made in his reports with those made at his deposition. *Hopton v. U.S. Steel Corp.*, 7 BLR 1-12 (1984). Specifically, Dr. Rosenberg stated in his initial report that "severe disabling COPD *does not* occur in relationship to coal mine dust exposure absent the presence of complicated CWP." (EX 7) (emphasis added). At his deposition, Dr. Rosenberg acknowledged making this statement but then backed away from this definitive assertion explaining that he was talking about a medical probability of greater than fifty percent and not absolutes. Dr. Rosenberg testified that he was "not sure" of the meaning of "reasonable degree of medical certainty" but later stated that the terms "medical probability" and "medical certainty" were the same thing. (EX 9, page 41). Dr. Rosenberg then agreed that simple pneumoconiosis could be disabling. (EX 9, page 35). In looking at the "four corners" of the initial report (EX 7), I find that Dr. Rosenberg's deposition comments are clearly inconsistent with the definitive statement contained within that report. Moreover, based on his deposition testimony, it is unclear what standard Dr. Rosenberg is employing at any given time in making his conclusions (i.e medical probability, medical certainty, or a reasonable degree of medical certainty?). Based on the foregoing, I accord less weight to the opinion of Dr. Rosenberg regarding the cause of death.

I accord less weight to the opinion of Dr. Bush who noted that significant CWP sufficient to cause impairment *or contribute to death* was not present based on the absence of abnormal arterial blood gases, no cor pulmonale, and the absence of consistent radiographic changes. (EX 8). However, according to Dr. Rosenberg, a highly qualified pulmonologist, the miner's COPD interfered with gas exchange as far back as 1989. (EX 9). Moreover, cor pulmonale was diagnosed by the miner's treating physician, Dr. Rasmussen as far back as 1997. (DX 14). As noted previously, I find that Dr. Rasmussen, as the miner's treating physician, was in a unique position to render an opinion in this matter. Therefore, without the benefit of an autopsy to confirm to the contrary, I give great weight to Dr. Rasmussen's opinion that the miner suffered from cor pulmonale. Accordingly, I find that basis of Dr. Bush's opinion regarding the cause of death is not well-documented and as such shall be accorded less weight. *Clark, supra.*

I accord less weight to the opinion of Dr. Caffrey as well. He noted that coal dust did not cause disability prior to the miner's death and did not hasten the miner's death. He then stated that if CWP were present, it would be so mild that it would not "by itself" cause a pulmonary disability. (EX 6). There is a fundamental flaw in the reasoning of Dr. Caffrey regarding this issue. Contrary to what he suggested, there is no requirement that coal dust exposure be the primary cause of a pulmonary disability in order to qualify as a factor in hastening the miner's death. Dr. Caffrey acknowledged in an earlier report that simple CWP could cause minor abnormalities in lung function. (EX 1). I found earlier that coal worker's pneumoconiosis was established in this matter. Perhaps in an otherwise healthy individual, the effects of mild simple CWP on a patient would be minor. However, I find that Dr. Caffrey failed to consider whether a minor abnormality in lung function due to simple CWP, *in a patient suffering from the effects of prolonged, heavy smoking and lung cancer*, could have hastened the miner's death in any way. Because Dr. Caffrey does not address this possibility in the proper context, I find his opinion is not well-reasoned and accord his opinion less weight. *Clark, supra*.

I accord less weight to the opinion of Dr. Castle. He opined the miner had significant COPD made up of a "significant asthmatic process" with some degree of emphysema. He agreed that coal dust exposure could be related to centriacinar emphysema in a statistically significant way and that it was statistically possible the miner had some minimal degree of emphysema that was due to coal mine dust exposure. However, Dr. Castle opined that this emphysema would not cause any clinically "significant" abnormalities. (EX 10). The fact that Dr. Castle chose to use the word "significant" indicates that the emphysema due to coal mine dust exposure could cause *some* abnormality or lung dysfunction. Perhaps in an otherwise healthy patient, this minimal emphysema would not have any significant clinical effect. However, I find that Dr. Castle failed to adequately explain how, in a patient with a history of prolonged heavy smoking and lung cancer, this minimal emphysema could have played no role in the miner's severe respiratory impairment that prevented him from having surgery to remove the malignant tumor at an early stage. Moreover, Dr. Castle did not adequately explain why coal mine dust exposure was eliminated as a possible cause of the miner's significant asthmatic process. For these reasons, I find Dr. Castle's opinion is not well-reasoned and as such will be accorded less weight. *Clark, supra*.

I accord less weight to the opinion of Dr. Naeye. Dr. Naeye opined the miner suffered from chronic bronchitis and mild emphysema. He added that if CWP were present it would be too mild to cause clinically significant abnormalities in lung function and could not have hastened death. (EX 6). Dr. Naeye noted that smoking had x4.5 greater influence in causing airways obstruction than coal mine dust exposure. Assuming this to be true, Dr. Naeye does not rule out the contribution of coal mine dust to the miner's significant obstructive impairment, he only minimizes its contribution relative to the effects of smoking. Dr. Naeye also noted that smoking, not coal dust exposure, was the *major* cause of pulmonary disability and death in United States coal mine workers. Dr. Naeye also noted that coal mine dust played "no role or only a minor role" in the genesis of centrilobular emphysema in the miner. (EX 11). Assuming coal mine dust exposure, in the instant case, is not the major cause of death, or is the major cause of the miner's emphysema, or is the major cause of the severe obstructive impairment, I find that Dr. Naeye does not adequately explain how even the acknowledged "minor" effects of coal mine dust exposure would not hasten death in a patient suffering from the effects of prolonged heavy smoking and advanced lung cancer. For these reasons, I find that the opinion of Dr. Naeye is not well-reasoned and is accordingly less persuasive than the opinion of Dr. Cohen on this issue and thereby I accord his opinion less weight. *Clark, supra.*

I accord less weight to the opinion of Dr. Zaldivar. Dr. Zaldivar noted the presence of asthma and mild emphysema due to smoking. He noted that emphysema was an added factor to the miner's death. I find that Dr. Zaldivar did not adequately explain how he was able to eliminate coal mine dust exposure as a possible cause or factor in the miner's asthma and in the development of emphysema. Dr. Zaldivar does not discuss the possible effects of coal mine dust in relationship to the miner's partially reversible obstructive defect. Accordingly, I find that the opinion of Dr. Zaldivar is not well-reasoned and is not well-documented and is less persuasive than the opinion of Dr. Cohen. Therefore, I accord his opinion less weight. *Clark, supra.*

Lastly, I accord less weight to the opinion of Dr. Morgan who premised his conclusions on the notion that the miner was not exposed to any harmful coal mine dust or silica. As noted previously, this assertion has no basis in fact and is contrary to all of the relevant evidence in the record. Therefore, I find his conclusions that coal mine dust exposure played no role in the development of any disability to have little merit. *Goss, supra.*

In summary, based on the foregoing, I find that the highly persuasive opinion of Dr. Cohen, i.e. that the miner's CWP hastened his death, outweighs the contrary medical opinion evidence of record. Accordingly, Claimant has established that the miner's death was due to pneumoconiosis pursuant to § 718.205(c)(2).

Date of Entitlement to Benefits

Since the miner died on May 5, 2000, Claimant is entitled to receive augmented benefits, as his surviving spouse, commencing as of May 1, 2000. See 20 C.F.R. § 725.212 and § 725.213(a).

Attorney's Fees

No award of attorney's fees for services to the Claimant is made herein since no application has been received. Thirty days are hereby allowed to Claimant's counsel for the submission of such application. His attention is directed to 20 C.F.R. §§ 725.365 and 725.366 of the regulations. A service sheet showing that service has been made upon all parties, including the Claimant, must accompany the application. Parties have ten days following receipt of such application within which to file any objections. The Act prohibits the charging of a fee in the absence of an approved application.

ORDER

The claim of Martha C. Williams, as surviving spouse of Alexander Williams, for black lung benefits under the Act is hereby GRANTED, and

It is hereby ORDERED that Consolidation Coal Company, the Responsible Operator, shall pay to the Claimant, Martha C. Williams, all augmented benefits to which she is entitled under the Act, commencing as of May 1, 2000.

A

MICHAEL P. LESNIAK
Administrative Law Judge

NOTICE OF APPEAL RIGHTS. Pursuant to 20 C.F.R. Section 725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 days from the date this Decision and Order was filed in the office of the District Director, by filing a notice of appeal with the *Benefits Review Board at P.O. Box 37601, Washington, DC 20013-7601*. A copy of a notice of appeal must also be served on Donald S. Shire, Esq. Associate Solicitor for Black Lung Benefits. His address is Frances Perkins Building, Room N-2117, 200 Constitution Avenue, NW, Washington, D.C. 20210.